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ANISEIKONIA

WALTER B. LANCASTER, M.D.

BOSTON

Aniseikonia is a term I coined for a difference in size of the optical images of the two eyes. By the optical image is meant not the retinal image but that which reaches consciousness as a perception. This definition leaves unanswered the question of where the cause of the difference is to be found, whether in the refraction mechanism or in histologic differences in the sensory elements of the retina or in the structures still higher up.

CONSEQUENCES

Aniseikonia throws a burden on what I am fond of calling "the neuromuscular mechanism for binocular vision." In some way the eyes must compensate for the aniseikonia, and in ordinary cases they do, so that binocular vision goes on satisfactorily. However, the amplitude of adjustment in compensation for aniseikonia is limited. Compare this amplitude of adjustment with other amplitudes of adjustment, viz., the amplitude of accommodation which compensates for certain common errors of refraction or the amplitude of fusion which compensates for heterophoria.

Whenever any one of these mechanisms of compensation shows signs of being overtaxed, symptoms occur. The symptoms which arise from decompensation in the mechanisms of ocular adjustment are singularly similar. It is not usually easy to tell from the symptoms, because they are the general symptoms of eyestrain, whether the accommodation or the fusion faculty or the unknown mechanism which cares for aniseikonia is being overtaxed. The fact that this mechanism is quite unknown at present should not be regarded as cause for reproach, since even after many years there is a serious lack of agreement as to the mechanism of accommodation. This does not prevent one from knowing much that is important and invaluable in practice about

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The discussion on this paper, which was combined with that on a paper by Dr. Conrad Berens, appears in the transactions of this society in this issue of the ARCHIVES, page 1109.

accommodation and how to deal with it. Considerable is known about aniseikonia considering the short time it has been under investigation and the limits in the number and resources of the workers whose leader was not an ophthalmologist, not even an optometrist, but an artist before he became a physiologist of the first rank by concentrating on these visual problems.

CAUSES

The most obvious cause of aniseikonia is anisometropia and the wearing of glasses of different magnifying power for the two eyes. An everyday problem in the practice of the ophthalmologist is how to deal with anisometropia. Small differences are easily managed by most patients, but few can take care of large differences.

While the fathers of ophthalmology knew about the troubles caused by anisometropia, they were not agreed as to the explanation. Donders and many others thought the unequal images important. Hess and others thought that the prismatic effect of looking through the periphery of the lenses was sufficient to tax the fusion faculty.

The next most obvious cause of aniseikonia is asymmetric convergence. When an object is brought near to the eyes, its image increases in size. If it is in the midline so that the eyes converge symmetrically on it, the increase is equal in the two eyes; but if the object is far to the right or left, it is obviously not equally near each eye but much nearer one eye. The retinal image in that eye is therefore larger, as was pointed out over two hundred years ago by Desaguliers. As in the case of aniseikonia due to anisometropia, so in that due to asymmetric convergence the eyes are able to compensate in some way for the aniseikonia and to carry on without trouble.

In some cases no well known cause exists to account for the aniseikonia. In such cases also the eyes are able to compensate for small or moderate amounts.

What do the eyes do when faced with a large amount of aniseikonia? When the task of compensation for any of the defects of binocular vision is too great for the amplitude of adjustment, the exit is by suppression and so by avoidance of binocular vision, with escape from eyestrain at the price of strabismus.

Given a case of strabismus, the question should arise: To what feature of the neuromuscular mechanism for binocular vision is it due? Is it perhaps due to the fact that the amplitude of fusion is inadequate to overcome a tendency to deviation from some cause, or is the relative accommodation inadequate to overcome a hypermetropia which is easily overcome by the total accommodation (which of course requires convergence for its full activity)?

In an uncertain percentage of cases the cause will be found to be aniseikonia of too great a degree. How common this is is not known, because it is not easy to measure aniseikonia in cases of strabismus. The ophthalmoeikonometer is designed only to test patients with binocular vision and fairly good vision in each eye. There is good reason to suspect that aniseikonia is a fairly common factor of importance in the causation of strabismus.

The consequences of aniseikonia may be considered from two points of view: (1) how it affects seeing and (2) what symptoms it causes.

The horopter will serve as an illustration of its effect on seeing. This is well demonstrated by the tilting plane. The significance of the experiment with the tilting plane is that it shows how aniseikonia can affect seeing, perspective and space perception, and therefore orientation.

There are many possible applications of the use of eikonic lenses, as for example, for aviators in landing. An ordinary person in viewing surfaces (landscapes) may have obscure difficulties in adjustments that would be helped by the wearing of eikonic lenses.

Such cases are not, in my opinion, very numerous.

Much harm has been done to the cause of aniseikonia by exaggerated claims for its cure. This is natural enough when a new discovery is made. The relief of patients for the first time by eikonic lenses is a thrill to the discoverer which must excuse enthusiasm. Indeed, lack of enthusiasm under the circumstances would be deplorable.

I have already mentioned clinical consequences of aniseikonia—eyestrain in some of its protean manifestations.

There have been various objections or criticisms to the claim that aniseikonia is a factor in binocular vision of sufficient importance to play a part in the practical treatment of eyestrain. I will mention three:

1. The benefit alleged to follow the use of eikonic lenses is really due to suggestion—a form of psychotherapy.

It is, of course, true that psychotherapy accounts for much of the benefit obtained in the treatment of eyestrain by spherocylindric lenses and by prisms as prescribed for astigmatism and heterophoria. No one with much experience and an open mind can fail to have seen cases illustrating this. The treatment of aniseikonia is no exception. On the contrary, the novelty, the prolonged and impressive examination, the "whoopee," if one likes to call it that, tend to impress the patient, who, by the nature of the condition (asthenopia), is more neurotic than the average patient. Hence the psychic factor plays a large part. Doubtless, more than one examiner has failed to allow for this in cases he has reported. Not to recognize the importance of this factor in esti-

mating the value of the treatment is a mark of lack of breadth of view and lack of adequate experience. It has always been emphasized at the Department of Research in Physiological Optics at Dartmouth Medical School.

On the other hand, to conclude that psychotherapy is the whole explanation, to say that since this case and that case are obviously of a psychic nature, all cases are of a psychic nature or are to be explained in some other way than that claimed by the investigators, is even more narrow, prejudiced and scientifically stupid. The only worse scientific sin is scientific dishonesty. To condemn a thing without a fair impartial hearing, without investigating it, is close to condemning it dishonestly.

2. A second criticism was advanced from the Wilmer Ophthalmological Institute and was elaborated by Friedenwald. The criticism is justified by a superficial study of the problem. Hess and others have pointed out the effect of the prismatic action of different lenses before the two eyes when the wearer looks through the periphery of the lenses. Eikonic lenses may have a considerable prismatic effect. They could act in two ways: 1. They could cause a strain by inducing a phoria for which the eyes have to compensate by fusion. 2. If there happened to exist a phoria of the right kind and degree, the eikonic lenses would correct it and so give comfort. Friedenwald, with his well known ingenuity and skill, builds up a plausible picture of anisophoria. By this he means phoria which differs in different directions of gaze, i. e., it is not concomitant. It is like the phoria produced by a slight paresis of some ocular muscle or muscles.

When the patient is being tested for aniseikonia, he is made to fix the eye on the central black disk, then to fix it on one after another of the four peripheral dots and stars. If there is anisophoria, when he looks, say, to the right, the dot and star will not be superimposed but separated in a typical way, the distance increasing the farther the eye is rotated in that direction. A lens with the properties of the eikonic lens could correct this.

There are various objections to this theory, but the following are easy to grasp and are convincing: (*a*) The tilting table effect cannot be explained by anisophoria but is readily explained by aniseikonia. (*b*) Instead of shifting the gaze from the center to the four peripheral dots, a different setup may be used. The essence of anisophoria is the difference in phoria in different directions of gaze. With this new setup, fixation is constant in the primary position. The observer looks at a suitable test object at the center of the screen seen binocularly. At about 200 mm. from this is a large object seen in the periphery of the retina. This is projected on the screen with polarized light as two images, one for each eye, one polarized vertically and the other horizon-

tally. The eyes seeing through polarized glasses see one image with each eye. If one image is displaced either by aniseikonia or in any other way, the distance from the center object which is being fixated to the peripheral object is not the same for each eye. The eyes cannot continue to fuse both the central object and at the same time the peripheral object; one must give way. One would expect that with the attention riveted on the object it would remain single and the peripheral object would become double. Instead, the peripheral images are fused and the center object is seen double. By measuring the separation by suitable means, the inequality (aniseikonia, if present) can be measured. Since the fixation throughout is on the center object there is no possibility of anisophoria affecting the result.

Incidentally, this importance of peripheral stimuli to fusion has been shown [1] to be so powerful as to make it impossible to maintain macular fusion under certain circumstances.

When I first saw the ophthalmoeikonometer, I raised the question: When the eyes shift from the central large black disk to the small dot and star, how does one know that fusion of the disk now in the periphery is maintained and that the eyes do not try instead to fuse the dot and star? I was told that trial had shown that in fact fusion is maintained by the stimulus in the periphery. Now there is astonishing proof of it.

3. The third objection was raised at the Howe Laboratory of Ophthalmology and elaborated by Ludvigh. It is based on the age-old observation that in asymmetric convergence the object is nearer one eye than the other, and so its retinal image is larger. This difference in size is greater than many of the differences which are claimed clinically to cause symptoms.

Aside from other answers to this criticism, the most convincing is the simple experiment of measuring the difference in size in asymmetric convergence when the object is the same distance from each eye. This is readily done with the Ames haploscope.

One eye looks at a circle and the other at a square the size of which equals the diameter of the circle. When they are superimposed, the circle would be tangent to the four sides of the square. If one is larger or smaller than the other, the difference would be easy to recognize and easy to measure by noting how much the smaller image needs to be moved nearer the eye to become equal to the size of the other image.

The astonishing fact is found that in asymmetric convergence there is an automatic increase in the size of the optical image of the adduct-

1. Burian, H.: Fusional Movements: Role of Peripheral Retinal Stimuli, Tr. Sect. Ophth., A. M. A., 1938, to be published.

ing eye. This equals, and therefore compensates perfectly for, the difference in the size of the retinal images due to the difference in distance as seen (without the haploscope) in ordinary vision.

The way the progressive development of the investigation has solved many of the objections raised by both friendly and hostile critics is one of the most impressive reasons for confidence in the Dartmouth Eye Institute.

Time will not permit an enumeration of the many mathematical, experimental, theoretic and practical clinical investigations made by Ames and his staff. Considering the novelty of the problems and the necessity of gathering and training a group of workers capable of supplementing each other's deficiencies and of cooperating with the musketeers' spirit of "all for one and one for all," and also the necessity of conceiving new methods of study, of designing and constructing new apparatus, of designing new types of lenses and enlisting the cooperation of the manufacturing optician in making them, of applying clinically the discoveries brought to light in the laboratory, which requires training technicians in the use of the apparatus, of devising new ways of approach to the problem in the effort to clarify its obviously numerous obscure and puzzling features, some of which have been detected by critics but many more of which have been seen and faced by the workers themselves—considering all these and many other difficulties which have been or are being overcome, the achievement in the few years that Ames has been working, having started literally from the beginning, is amazing. It commands the enthusiastic support of all who have the interests of the progress in ophthalmology at heart. To throw obstacles in its way simply because, like all pioneer work, it is not flawless is most unbecoming, not to say unfair. Constructive criticism is always in order and always welcome.

PHYSIOLOGIC AND CLINICAL OPHTHALMOLOGIC PROBLEMS IN RELATION TO INDIVIDUAL VARIABILITY

A. BRÜCKNER
BASEL, SWITZERLAND

(Concluded from page 756)

LIGHT AND DARK ADAPTATION

As an example of the general adaptation of the eye mentioned at the beginning of this article, observation shows that the eye is able to adapt itself to very different degrees of intensity of light.[75] The investigations of the twentieth century have increased knowledge concerning light and dark adaptation greatly, although many questions are not yet solved.

The method of measuring the degree of general adaptation consists in the observation of an uncolored field of a definite size which can be illuminated by different intensities of light. The range of these intensities is great. Many forms of apparatus have been constructed; one of the best seems to me to be that of Nagel.[48] The intensity measurable can be varied from unity to 8,000,000.

The subject first studied is normal and pathologic dark adaptation, although light adaptation also shows individual differences and perhaps pathologic deficiencies. But at present not enough is known about it, especially as the measurement of increasing light adaptation presents greater difficulties than that of dark adaptation only.[76]

Former investigations were confined to the determination of the relative thresholds, without measurement of the absolute intensity of

75. A non-neutral (not absolutely white) illumination, such as is given by an ordinary artificial source of light with its insufficiency of short wavelengths, causes a general alteration of the sensitivity of the eye. Usually this is noticed only when the phenomenon is very strong. I had an experience in connection with this problem many years ago. Awakening in the morning in a strange room, I saw all things bluish green; even my hands were the color of a corpse. The explanation is that while I slept the sun had shone through the closed lids into my eyes for a long time. Therefore, the eye was considerably exhausted for yellowish red, the color perceived by closed eyes, and the negative phase produced a hypersensitivity to the bluish green color and an insensitivity to red and yellowish color. After about two hours normal sensitiveness returned. The phenomenon is similar to the so-called erythropsia after snow dazzle.

76. Lohmann, W.: Ueber Helladaptation, Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. (Abt. 2) **54**:161, 1922.

light in the observation field. From such study in many cases of night blindness sufficient information may be obtained for practical purposes. But to detect slight differences of dark adaptation it is necessary to measure the intensity exactly, in lux or foot candles. Since it was desired to be more exact, the results obtained for a normal observer were compared with those for a person with a disturbance of dark adaptation, on the assumption, which is not justified, that all normal persons have the same course and degree of dark adaptation.

There are some difficulties or sources of error which must be avoided.

At first it seems to be easy to determine whether one sees the field of the adaptometer or not. But in the diminution of the thresholds some troublesome factors arise. The problem is to determine the difference between the subjective sensation of gray from one's eye which arises when an absolutely dark field is observed and a sensation only noticeably brighter produced by an objective stimulus. This is often difficult, because after a stay of about ten minutes in the dark room, as an expression of the change in the visual substance entoptic phenomena arise, such as bright clouds and flickering. These sources of error may be partly eliminated by experience and attention.

The width of the pupil must be rendered constant by artificial mydriasis. Although in the darkness the pupil dilates automatically, differences occur according to age; older persons have narrower pupils than younger ones. The amount of the light entering the eye naturally depends on the size of the pupil. The formula πr^2 shows that when the diameters of two pupils have the ratio 2:8 their areas have a ratio of 3:48; hence the intensity of light on the retina with these pupils is of the ratio 1:16. The thresholds, therefore, must vary if the pupil does not dilate owing to illness, for instance, in tabes dorsalis. The suggestion made by some writers that the reduction of adaptation in this disease is evidence of the central regulation of dark adaptation is not convincing, since this source of error was not always excluded. For the same reason the suggestion of Lasareff[77] is not correct. He stated that with increasing age the adaptation to light was found to diminish more and more. Therefore, Lasareff found by extrapolation of the curve that at about 150 years the adaptation should be zero and suggested that 150 years should be the moment of physiologic death! Besides the doubts which must naturally arise about extrapolation in general, this suggestion is certainly false, since he neglected the size of the pupil, which as a result of the increasing rigidity of the tissue of the iris becomes narrower with increasing age. After

77. Lasareff, P.: Sur le changement de la sensibilité au cours de la vision périphérique avec l'âge de l'homme, Riv. di biol. **10**:617, 1928.

artificial dilation of the pupil, such as that used by Hans Karl Müller [78] and Geneviève Matthey [79] in their experiments, carried out in Basel, a diminution of dark adaptation dependent on age is not to be found.

A further factor influencing the result is the state of the visual organ at the beginning of the experiment, depending on the duration and the degree of the previous illumination. Recent observers have illuminated the eye by means of a brightly illuminated white sphere (3,000 lux vertical to barium sulfate) for ten minutes after a stay of thirty minutes in a dark room.[80] One may then suppose that the conditions of general adaptation are equal in all cases, especially if during the process of light adaptation the eyes are moved in all directions.

It is known that a retinal zone of some excentricity—about 20 degrees—to the macula adapts best to darkness. One often takes this into consideration, using a small red point, situated above or beside the field of the adaptometer, for the fixation of the eye.

All these precautions were taken in researches carried out by Hans Karl Müller [78] and his pupil Geneviève Matthey [79] in the ophthalmologic clinic in Basel.

In the measurement of the threshold value of the illumination of the field, a stimulus a little above the threshold was first given. This was diminished and the moment of disappearance noticed. The values were taken every three minutes during a stay up to three fourths of an hour in the dark room. A curve was then constructed in a manner differing from the usual procedure.

Nagel [48] and Piper [81] did not plot the values for thresholds themselves but used their reciprocal values and treated these as a measure of the sensitiveness of the eye. In the first few minutes after light adaptation the thresholds are very high, but they quickly decrease. Their reciprocal values are very small. For this reason a curve, as shown in figure 39, taken from Piper, must produce the impression that during the first six to eight minutes the dark adaptation increases very slowly. That is not true, as any one who enters a dark room will observe.

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78. Müller, H. K.: (*a*) Ueber den Einfluss verschieden langer Vorbelichtung auf die Dunkeladaptation und auf die Fehlergrösse der Schwellenreizbestimmung während der Dunkelanpassung, Arch. f. Ophth. **125**:624, 1931; (*b*) Zur Darstellung des Dunkeladaptationsverlaufes in Kurvenform für klinische Untersuchungen, ibid. **125**:614, 1931.

79. Matthey, G.: Eine "Standardkurve" der Dunkeladaptation für klinische Untersuchungen, Arch. f. Ophth. **129**:275, 1932.

80. Hertel: Untersuchungen des Lichtsinnes mit einem Kugeladaptometer, ein Weg zu seiner Standardisierung, in Concilium Ophthalmologicum, Leiden, Netherlands, Eduard Fjdo, 1929, vol. 4.

81. Piper, H.: Ueber Dunkeladaptation, Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. (Abt. 2) **31**:161, 1903.

If the intensity of the thresholds themselves or their logarithms are plotted, the curve falls quickly in the first minutes, as is shown in figure 40.

In passing, some experiments may be mentioned which were intended to exclude the influence of a longer duration of the stimulus in causing a local adaptation. Momentary stimuli were used which, according to the so-called method of mean variation, were presented by a camera shutter, as shown in figure 41. The value of the thresholds depends on the time of exposure, as in using colored stimuli for the light-adapted eye. But this method is inconvenient, since it requires a number of single experiments, which fatigue the observer. Nevertheless, the results demonstrate that with the increase in the time of exposure the thresholds diminish. Therefore, it may be concluded that after exposure to a stimulus of a not too great duration the thresholds will be lower.

It was obvious that it would be possible to obtain a curve demonstrating the normal course of dark adaptation only by ascertaining the

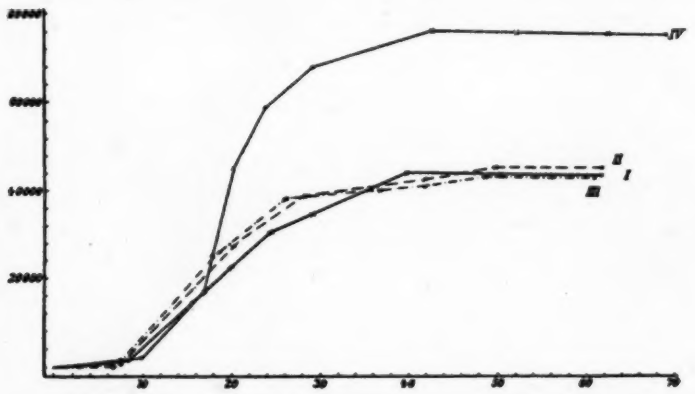

Fig. 39.—*IV* indicates binocular adaptation and observation; *I*, right eye observes, both eyes adapted; *II*, right eye dark, left eye light adapted, only right eye observes; *III*, right eye dark, left eye light adapted, both eyes observe. (After Piper.[81])

averages from a great number of observers. Matthey examined 54 normal persons by monocular vision.

Figure 42 shows the standard curve for normal persons from the measurements made by Miss Matthey. There is no point in calculating the curve of variation to the other side, since the values would to a great extent lie below zero. I think that these curves, although obtained on only 54 persons, demonstrate the individual variability and also show whether dark adaptation is normal or not. When a curve lies outside, especially higher than the broken line, the dark adaptation is to be considered pathologic.

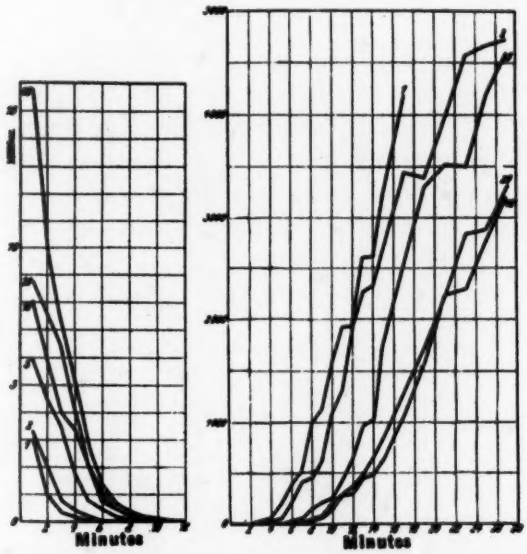

Fig. 40.—Adaptation curves (left chart) after preillumination of varying duration, represented by the irritation thresholds, which are given in millilux (average value of 10 single experiments). The numbers on the curves correspond to the duration of preillumination in minutes. (After H. K. Müller.[78a]) Adaptation curves (right chart) after preillumination of varying duration, represented by the reciprocal values of the irritation thresholds (average values from 10 single values, except with forty minutes). The numbers on the curves correspond to the duration of preillumination in minutes. (After H. K. Müller.[78a])

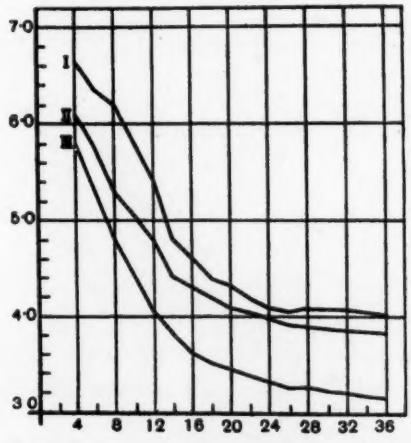

Fig. 41.—Curve of the threshold values for exposures of one-fiftieth second (*I*), one-tenth second (*II*) and with time illumination (vanishing threshold, *III*), with reference to a relative unit, represented by logarithms. (After Brückner.[64]) Abscissas indicate duration of dark adaptation in minutes.

Recently a standard curve has been published by Ferree, Rand and Stoll.[82] It is based on over 200 single curves. Unfortunately, the curve extends only to the twentieth minute after the beginning of adaptation, although important differences may occur after this time.

Wölfflin [83] suggested that persons with marked pigmentation of the fundus have better dark adaptation than those with little pigmentation. Certainly the latter are often more affected by strong illumination; they have headache, are dazzled, and so on, apparently because the damping of the light in the retina is too slight as a result of lack of pigmentation of the fundus oculi. But Miss Matthey did not find any differences in eyes with these two types of pigmentation (fig. 43).

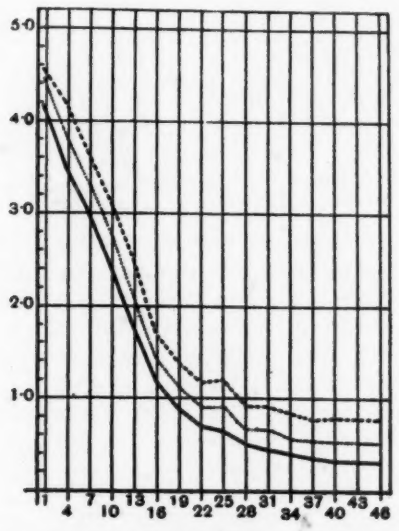

Fig. 42.—Standard curve of dark adaptation. The continuous line corresponds to the averages expressed as logarithms with a base of 10. The broken line shows the upper limit of scatter found by addition of the logarithms of the mean values and three times the amount of mean error of the single observation (3σ), while the dotted line represents the sum of the average and the single value of errors. (After Matthey.[79])

The dependence of the dark adaptation on age, suggested by many authors and recently by Ferree, Rand and Stoll, has already been mentioned (fig. 44).

There is also another result of some importance. If the observation is begun after there is good adaptation to light, in the first five

82. Ferree, C. E.; Rand, G., and Stoll, M. R.: Critical Values for the Light Minimum and for the Amount and Rapidity of Dark Adaptation, Brit. J. Ophth. **18**:673, 1934.

83. Wölfflin, E.: Einfluss des Lebensalters auf den Lichtsinn bei dunkeladaptiertem Auge, Arch. f. Ophth. **61**:524, 1905.

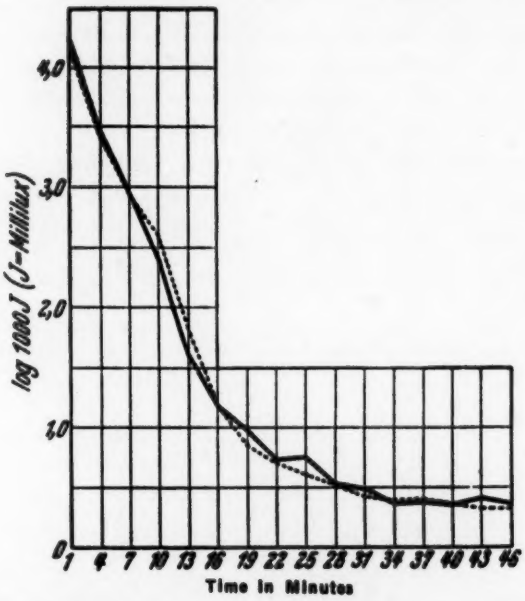

Fig. 43.—Dark adaptation. The unbroken curve corresponds to the average values of the intensities of stimulation thresholds in persons with a relatively slight pigmentation of the ocular fundi; the dotted curve is for persons with a relatively marked pigmentation of the fundus oculi. (After Matthey.[79])

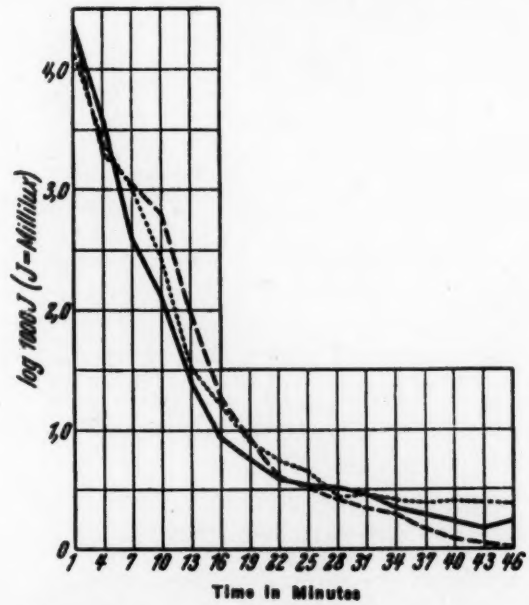

Fig. 44.—Dark adaptation. The dotted curve corresponds to the average values of the intensities of stimulation thresholds in persons of 11 to 17 years; the broken one is for persons of 18 to 39 years and the unbroken one for persons of 45 to 59 years. (After Matthey.[79])

to seven minutes the daylight apparatus functions alone; then follows a period during which both the dark and the daylight apparatus will be stimulated by the light, and finally a period during which only the dark apparatus reacts to the low stimuli.

This consideration leads to the theoretic suggestion of four possible types of adaptation:

1. A type with fairly high thresholds both at the beginning and at the end, corresponding to an imperfect working of both the light and the dark apparatus.

2. A type with relatively high thresholds at the beginning and fairly low thresholds at the end of the course. This corresponds to a poorly

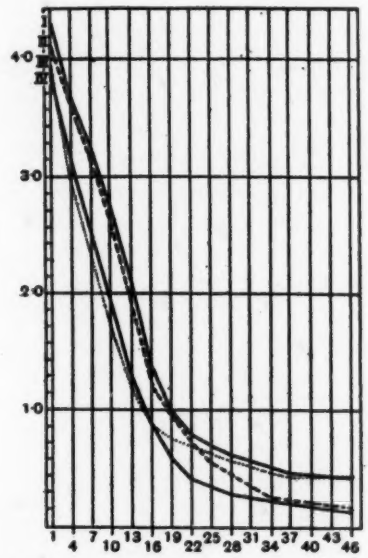

Fig. 45.—Course of adaptation curves in the four different types of adaptation which can be distinguished. (After Matthey.[79])

functioning daylight apparatus with a dark apparatus which functions well.

3. Inversely, a type with relatively low thresholds at the beginning and relatively high thresholds at the end.

4. The best type, with low thresholds throughout, corresponding to a good functioning of both the light and the dark apparatus.

The curves in figure 45 show these four types of adaptation. If the differences, especially at the beginning of the adaptation, do not seem to be large, it must be remembered that the curves represent the logarithmic values of the stimuli, and that the real differences are large.

If the occurrence of these four types is confirmed by further observations, this will support the so-called duplicity theory. While Hering [84] expressed the opinion that the visual substance was uniform, the zoologist Max Schultze [85] in 1866 tried to prove by comparative anatomic studies that cones predominated in the retina of the daylight animals and rods in that of the night animals. This theory was amplified by Parinaud [86] in Paris and von Kries in Freiburg.[87] According to the duplicity theory, the light apparatus lies in the cones and the dark apparatus in the rods of the retina (fig. 46). The latter alone contain the visual purple in their external limbs. The absorption which takes place when the different wavelengths of light pass through a solution of visual purple accords satisfactorily with the curve of the brightness of the spectrum at low intensity observed by the dark-adapted eye; under these conditions the eye does not see any color, but everything seems whitish gray or black. The curve coincides with that of the bleaching

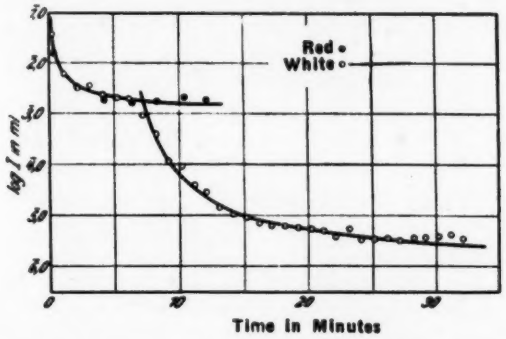

Fig. 46.—The white circles correspond to the logarithms of the stimulation thresholds in an adaptation experiment. There is a definite turning point between the fifth and the tenth minute. The black circles are the logarithms of the stimulation thresholds in another adaptation experiment, in which only the cones of the fovea have been excited. The curves correspond to the values which Hecht calculated on the basis of his adaptation formula. Hecht has, for the mathematical analysis, separated the adaptation curve into two independent curves: One comprises the course of adaptation up to the turning point and illustrates the dark adaptation of the light apparatus; the other corresponds to the adaptation of the scotopic apparatus. (After Hecht, S.: Ergebn. d. Physiol. **32**:282, 1931.)

84. Hering,[5] 1920, vol. 2.

85. Schultze, M.: Zur Anatomie und Physiologie der Retina, Arch. f. mikr. Anat. **2**:175, 1866.

86. Parinaud, H.: La vision, Paris, Octave Doin, 1898.

87. von Kries: Zur Theorie des Tages- und Dämmerungssehens, in Bethe A., and others: Handbuch der normalen und pathologischen Physiologie, Berlin, Julius Springer, 1929, vol. 12, pt. 1, p. 678.

values of spectral lights for the visual purple (fig. 47). This fact suggests that the latter is the substance the disintegration of which stimulates the rods in the retina.

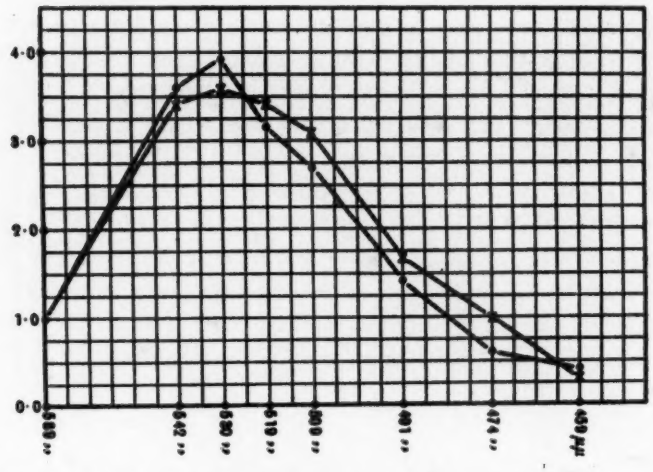

Fig. 47.—The X and dash line indicates the bleaching values of spectral lights for frog's visual purple, and the dash and dot line, the human achromatic scotopic luminosity curve. The abscissas indicate the wavelengths of the prismatic spectrum of the Nernst light; the ordinates, an arbitrary scale. (After Trendelenburg: Ergebn. d. Psysiol. **11**:1, 1911.)

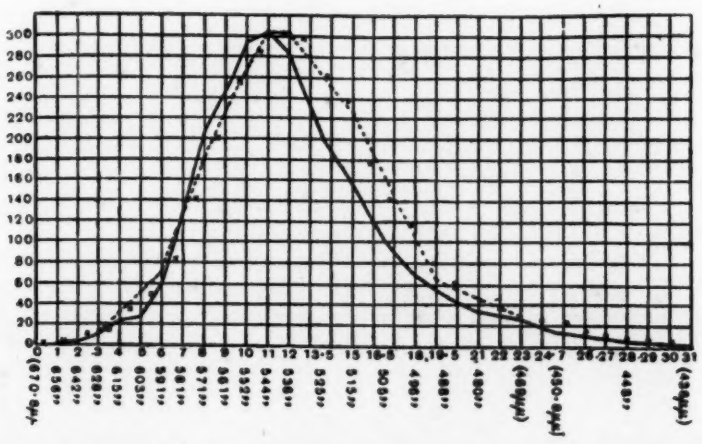

Fig. 48.—The continuous line indicates the achromatic scotopic luminosity curve of a deuteranope. The dotted line is the luminosity curve of a monochromat. The abscissas indicate wavelengths of the prismatic spectrum of gaslight; the ordinates, an arbitrary scale. (After von Kries, J.: Ueber Farbensysteme, Ztschr. f. Psychol. **13**:241, 1897.)

As mentioned before, the scotopic luminosity curve is the curve for persons with total congenital color blindness (figs. 48 and 14).

The observations of general adaptation hitherto described were all concerned with the results obtained with one eye. When both eyes are used, the binocular thresholds change when the dark adaptation has progressed, namely, after a stay of ten or twelve minutes in the dark room. The binocular threshold has then only half the value of the monocular (fig. 39). The statements of writers differ on this point. Nevertheless, there is a remarkable difference between the light-adapted eye and the dark-adapted eye. The summation of the sensations is not to be found with the light-adapted eye, even when stronger stimuli are used: The brightness of an uncolored field is almost the same with monocular as with binocular vision.

Meanwhile, new experiments dealing with this problem have been carried out by G. Schumacher [88] in the ophthalmologic clinic in Basel. As a stimulus this author used red light, which can be perceived only by the daylight apparatus. He found that only the dark apparatus has the faculty of summation of the impressions of both eyes.

The difference between the dark-adapted and the light-adapted eye is of theoretic interest. The summation of the stimuli as the correlative of the psychic sensations must be localized in the central part of the visual pathway. In all probability it is situated in the corpus geniculatum laterale or the visual cortex of the posterior lobe or even higher. In view of the influence of the state of adaptation on the summation, one must suppose that these central parts are also influenced by the general adaptation.

Starting from the duplicity theory, which is based on the presence of different retinal elements, it is not easy to understand how the change of excitability in the retina can influence the nerve centers. Although it has not been sufficiently demonstrated, one must suppose that, just as I have shown for local adaptation, the light-dark adaptation is not confined to the peripheral organ.

Some other facts point in the same direction. The entoptic visibility of the blindspot is especially distinct when the eyes are slightly dark adapted, for instance, in looking at the sky after sunset. Extreme degrees of dark or light adaptation are both unfavorable for this observation. Because the visibility of the blindspot, as already mentioned, is localized in the central parts of the visual pathway, it seems that the general adaptation influences these processes dependent on the contrast.

88. Schumacher, G.: Ueber das Verhalten der monokularen und binokularen Reizschwelle während der Dunkeladaptation des Tages- und Dämmerungsapparates, Acta ophth. **15**:5, 1937. This article gives references to other literature on this subject.

A little while ago Frey,[89] in Zurich, Switzerland, found centrifugal neural paths between central parts of the brain trunk and the retina. This fact also suggests, from the anatomic point of view, not only a central influence on the peripheral organ but the possibility of a reciprocal relation between the brain and the retina. In this way the importance of dark adaptation in the phenomena of contrast may be understood.

I shall now speak of the stimulation of the eye not by the adequate light stimulus but by the electric current. The results here differ if the thresholds of the light-adapted and of the dark-adapted eye are compared. Georg Elias Müller[90] found the same thresholds for both, but others[91] found the threshold for the dark-adapted eye about half that of the light-adapted eye. Certainly the results were obtained before chronaxia, first studied by Lapicque, was known. Therefore, Achelis and Merkulow,[92] who considered this point, were probably right when they stated that the irritability of the retina was equal in both cases. It seems to me, however, that this question is by no means fully elucidated. In all cases of electric stimulation the amount by which the threshold stimulus is diminished is of a totally different size from that observed when the specific light stimulus is used. The electric current must act on the visual substance at another part, perhaps, as some authors (for instance, Hecht) have suggested, in the optic nerve and not in the retina. In this respect it may be of interest to record that a summation of electric stimuli is apparently to be found only in the dark-adapted eye (table 4).

DISEASES ACCOMPANIED BY DISTURBANCES OF DARK ADAPTATION

I shall discuss briefly some diseases which are accompanied by disturbances of dark adaptation. This disturbance is called night blindness, or chicken blindness, as the Russians call it on the supposition that fowls go to sleep early in the evening because they cannot see at night.

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89. Frey, E.: Vergleichend-anatomische Untersuchungen über die basale optische Wurzel, die Commissura transversa Gudden und über eine Verbindung der Netzhaut mit dem vegetativen Gebiet im Hypothalamus durch eine "dorsale hypothalamische Wirzel" des Nervus opticus bei Amnioten, Schweiz. Arch. f. Neurol. u. Psychiat. **39**:255, 1937; **40**:69, 1937.

90. Müller, G. E.: Ueber die galvanischen Gesichtsempfindungen, Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. **14**:329, 1897.

91. Brückner, A., and Kirsch, R.: Ueber den Einfluss des Adaptationszustandes auf die Empfindlichkeit des Auges für galvanische Reizung, Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. (Abt. 2) **47**:46, 1912.

92. Achelis, J. D., and Merkulow, J.: Die elektrische Erregbarkeit des menschlichen Auges während der Dunkeladaptation, Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. (Abt. 2) **60**:95, 1929.

Those affected by night blindness cannot see sufficiently at low intensities and therefore are often disturbed when outdoors at night, especially in the country. During the World War, such persons, who in the cities did not perceive any incapacity, were unfit for military service, a great part of which was carried on after dark. I saw many

TABLE 4.—*Summation of Binocular Excitations by Electric Stimuli in the Light-Adapted and in the Dark-Adapted Eye of Two Observers (After Brückner and Kirsch* [91])

				Summation in	Average Summation in
Light	1. K.	16 +	24 —	40%	40.5%
	2. K.	16 +	33 —	33%	
	3. B.	25 +	38 —	40%	
	4. B.	39 +	40 —	49%	
Dark	5. K.	55 +	31 —	64%	75.5%
	6. K.	51 +	23 —	69%	
	7. B.	42 +	8 —	84%	
	8. B.	34 +	6 —	85%	

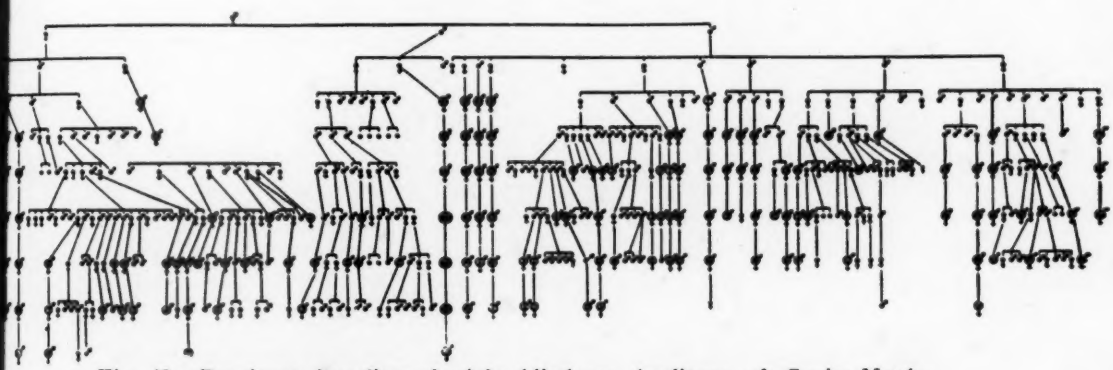

Fig. 49.—Dominant heredity of night blindness (pedigree of Cunier-Nettleship). ♂ indicates affected male; ♀, affected female; ♂, healthy male; ♀, healthy female; the circles containing numbers, the number of healthy offspring. (After Francheschetti, in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 1.)

of these men. They were sent back because their dark vision was insufficient. The degree of night blindness was often slight and the clinical state of the eye normal.

A good deal of night blindness is hereditary. The famous pedigree published by Cunier-Nettleship, which includes ten generations, is well known (fig. 49). The hereditary form is dominant. Curves of the adaptation made under the strict conditions mentioned in this article do not exist.

An acquired form of night blindness, with or without a condition of the conjunctiva known as xerosis, is well known to be caused by an insufficiency of vitamin A. In Denmark, during the war, night blindness was endemic because the population had to eat margarine,

which does not contain vitamin A. A similar condition is frequently encountered in Russia, as a result of fasting during Lent. Occasionally, it occurs also in Western Europe in the spring. The treatment is easy, because it is only necessary to give the patients vitamin A in the form of cod liver oil, fresh vegetables or carrots.

The disturbances of adaptation found in retinitis pigmentosa, a degeneration of the retina which is included in the so-called hereditary familial degeneration of the central nervous system, is known by all ophthalmologists. Types are found to vary from one generation to another. Unfortunately, so far there is no successful treatment for this disease.[93]

As a result of the anatomic alterations in the retina there are deficiencies of dark adaptation in high myopia. The stretching of the inner layer in the posterior part of the eyeball apparently affects the nutrition of the retina.

I shall discuss with a little more detail a special form of myopia found in the eyes of children. An investigation into this condition was carried out in the ophthalmologic clinic of Basel in the eyes of 22 young children.[94] Probably such myopia is congenital. A definite type of heredity was not to be found. The fundus oculi is relatively lightly pigmented at the posterior pole, but the region of the macula, on the other hand, is markedly pigmented. The visual acuity is always more or less diminished; in the majority of cases it is not more than a third of normal. The dark adaptation, as seen in figure 50, which shows the average values in the 22 cases in comparison with the standard curve of the normal eye, is not much higher, and after three fourths of an hour the thresholds are nearly the same. In respect to the evaluated curves, the thresholds at the beginning of the adaptation are decidedly higher than the normal.

These facts suggest that in this special form of myopia the daylight apparatus is in some way deficient and the dark apparatus is not impaired. Still more, this opinion seems to be supported by a single curve, such as that shown for instance in figure 51, which shows the relation of the upper limit of the variations of normal adaptation to the adaptation curve of the myopic child. During the first ten minutes the curve lies outside the limit of normal, but it is thereafter nearly normal.

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93. Investigations just published by E. M. Josephson and M. Freiberger (Carotene Therapy of Retinitis Pigmentosa, Nature, London **139**:155, 1937) seem to prove that the intramuscular injection of carotene diminishes the night blindness in the initial stages of retinitis pigmentosa.

94. Brückner, A., and Franceschetti, A.: Myopie im Kindesalter, Arch. f. Augenh. **105**:1, 1931.

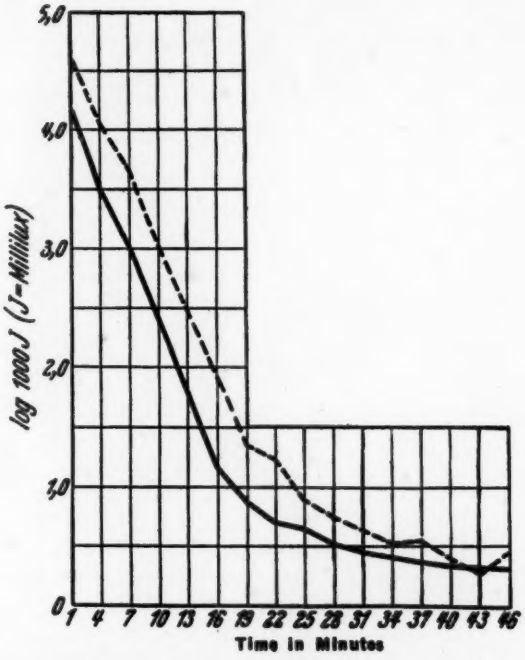

Fig. 50.—The continuous curve represents the "standard curve" of dark adaptation; the dash line corresponds to the average values of the intensities of the threshold stimuli in 22 children with myopia of the Brückner and Franceschetti type. (After Brückner and Franceschetti.[94])

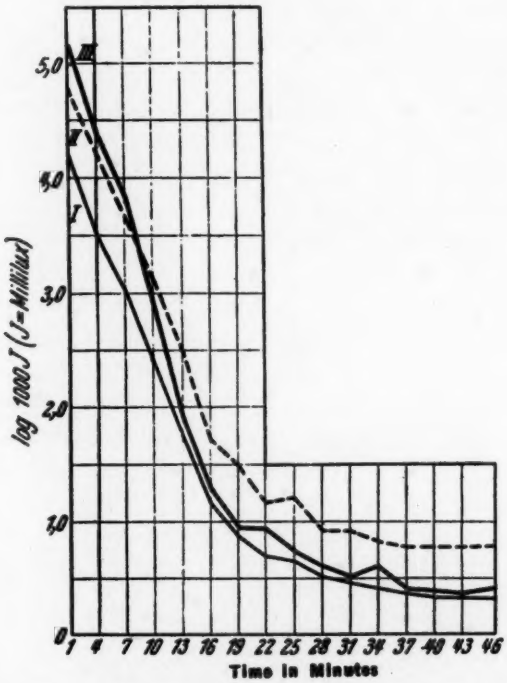

Fig. 51.—Curve *I* indicates "standard curve"; curve *II*, upper boundary of spread; curve *III*, adaptation curve of a child with myopia of the Brückner-Francheschetti type. (After Brückner and Francheschetti.[94])

CLINICAL METHODS

FOCAL ILLUMINATION

Although in individual clinical cases the estimation of the visual acuity is often difficult, the results obtained by intelligent persons with normal eyes agree sufficiently. The variation in the illumination of the test cards is of most importance. If this is too low or too high the visual acuity is less than with a moderate degree of illumination. Probably the curve of visual acuity is not a linear function of the intensity of the illumination according to the Weber-Fechner law but is, as Janisch [95] suggested, a so-called chain function (*Kettenfunktion*). The difficulty arises from the fact that alteration in the intensity of illumination also produces changes in the adaptation of the eye, and it takes some time for these changes to occur.

But I shall not discuss again this complicated subject, which helps one to understand the physiology or psychology of the procedure known as focal illumination. This consists in the concentration of light by means of a convex lens on that part of the eye which it is desired to examine exactly, e. g., a cornea with opacity. This is seen much more clearly by focal illumination than by simple diffuse light. If the Weber-Fechner law were valid for the relative intensities of illumination involved, there would be no improvement of discrimination.

One reason for the increase in visibility of the opacity is the slight degree of dark adaptation which takes place when this method is used in the dark room; but there is another, more physical, factor. According to Lord Rayleigh, the intensity of the light diffracted by the dispersed particles of a medium increases in proportion to the sixth power of the size of the dispersed particles. But this is valid only in the case of the colloidal dispersions from 0.1 to about 1 millimicron (fig. 52). If the dispersed particles are larger, the diffraction again decreases and the visible opacity diminishes.

Thus it is understood why in the intensive light of the slit lamp the normal cornea appears not clear but relucent. If the cornea has opacities, these may also be seen by daylight, because the degree of dispersion in them is larger than in a clear cornea. I suggest that it is in this and not in the rough anatomic structure of the scars in the cornea that the reason for the opaque appearance of these parts must be sought.

TONOMETRY

Generally speaking, the ophthalmologist makes use of the visual sense when examining his patients. The physician or the surgeon more frequently employs the sense of hearing or touch. Therefore, the

95. Janisch, E.: Das Exponentialgesetz als Grundlage einer vergleichenden Biologie, Berlin, Julius Springer, 1927.

ophthalmologist often feels lost if he cannot use his eyes, as in the case of a growth in the orbit or elsewhere.

But in the investigation of the intraocular pressure, the sense of touch is often used in feeling the eyeball through the lids. This method, first employed by Bowman,[96] is still used by many physicians, although the majority now use a so-called tonometer. By means of this instrument the estimation of the intraocular pressure is transferred from the sense of touch to that of vision. It is necessary only to observe the position of an indicator on a scale.

Although these instruments have also their sources of error, the measurement is thereby made much more exact than the mere touch. The absolute value of the intraocular pressure is not measured, chiefly because the rigidity of the cornea varies according to the age of the patient. Nevertheless, by the use of new curves, calculated by Hans

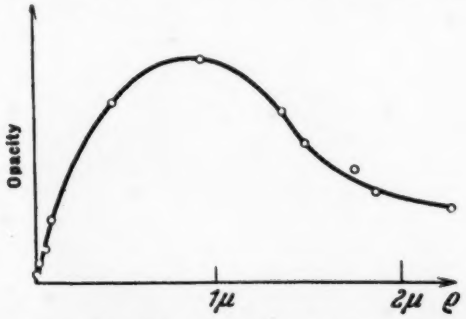

Fig. 52.—Dependence of the intensity of the dulness on the size of the particles; it increases to a maximum and then decreases again. (After Ostwald, C. W. W.: Kolloidwissenschaft, Elektrotechnik und heterogene Katalyse, Kolloidchem. Beihefte, 1930, vol. 32, nos. 1-4.)

Karl Müller,[97] this pressure may be estimated with sufficient accuracy (fig. 53).

It is interesting to determine the amount of error present when the physician estimates the tension in millimeters of mercury by the fingers and immediately before or after this determines the tension instrumentally. This estimation is more complicated than the original method of Bowman. He distinguished nine degrees: normal tension, tension doubtfully, just or distinctly increased or diminished, and the two extremes, with the eyeball hard as a stone or soft as pap.

In figure 54 the pressure determined by the tonometer is shown by the abscissas and the pressure determined by the fingers by the ordinates. *M*, the curve of averages, shows that from 10 to 30 mm.

96. Bowman, W.: On Glaucomatous Affections and Their Treatment by Iridectomy, Brit. M. J. **2**:377, 1862.

97. Müller, H. K.: (*a*) Augendruck und Lebensalter, Arch. f. Augenh. **105**: 504, 1932; (*b*) Ueber die Fehlergrösse der Augendruckmessung mit dem Tonometer von Schiötz, ibid. **105**:516, 1932.

the coincidence is satisfactory. But in the higher pressures the estimate by the fingers is always too low. In the spread allowed by one (σ) and three times the standard deviation (3σ) the error may be

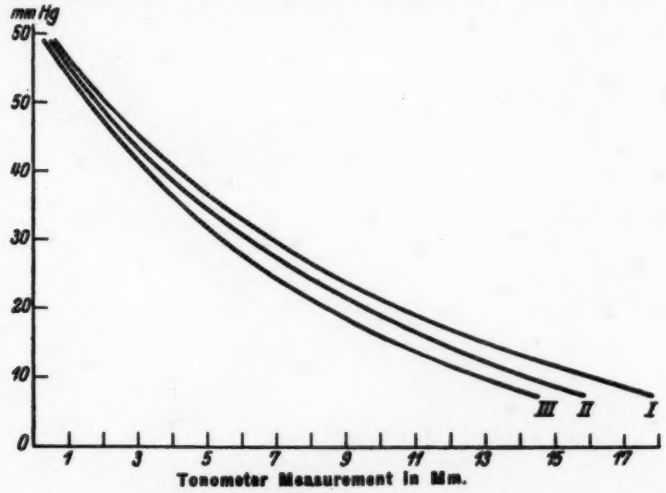

Fig. 53.—Tonometric gaging curves for the 7.5 weight (original Schiötz tomometer). Curve *I* is for children between 1 and 10 years of age; curve *II*, for persons between 20 and 44 years; curve *III*, for persons between 45 and 90 years. (After H. K. Müller.[97a])

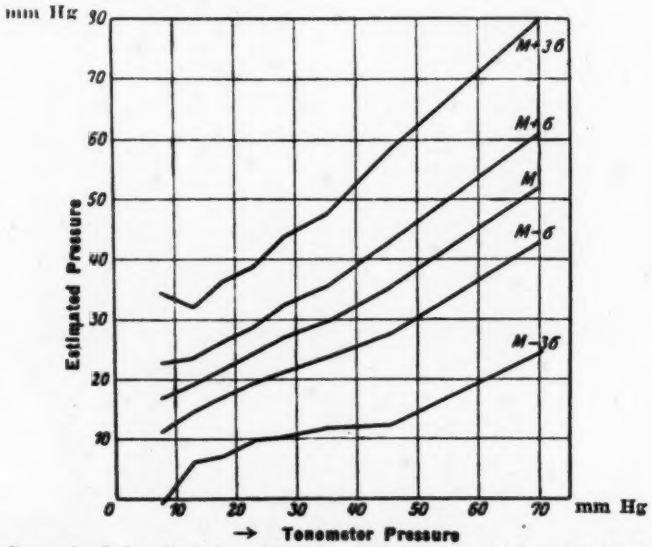

Fig. 54.—Spread of the digital estimation of pressure. (After H. K. Müller.[97b])

considered excessive. For instance, a tension of 60 mm. may be estimated as 20 mm. or one of 25 mm. as 40 mm. of mercury. If a pressure of 25 mm. is considered the upper normal limit, the results show that it is almost certain that the pressure lies within the normal limits when the eye is estimated as very soft.

Even in determining only the difference between the two eyes, the errors are important. The experience of my colleagues and myself has shown that in 36 per cent of cases the difference was not correctly estimated.

These results show clearly that it is impossible to rely on the fingers as a method of estimating tension in glaucoma.

SENSITIVENESS OF THE CORNEA

In order to obtain knowledge of the state of the sense organs of the skin in disease it has been necessary to obtain exact values for the stimulus applied in testing that sensation. In the skin one distinguishes different qualities of sensation: those of touch, pain, cold and warmth. Knowledge of the function of these senses has been furthered by Max von Frey,[98] Goldscheider,[99] Blix [100] and others. Von Frey used hairs (fig. 55) of different stimulus value, which he determined from the relation between the area of the hair and the weight it was able to support. This pressure value, as von Frey called it, is determined by

Fig. 55.—Stimulus hair. (After von Frey.[98a])

the formula $\frac{\text{grams supported}}{\text{area of hair in sq. mm.}}$, which gives a figure of greatest importance for measuring tactile thresholds in skin.

The same method has also been used in recent times by ophthalmologists, for instance, Marx,[101] Stüdemann,[102] and Pflimlin.[103] It was used especially to determine the thresholds of the cornea.

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98. von Frey, M.: (*a*) Beiträge zur Physiologie des Schmerzsinnes, Ber. u. d. Verhandl. d. k. sächs. Gesellsch. d. Wissensch. zu Leipzig, math.-phys. Kl. **46**:187, 1894; (*b*) Untersuchungen über die Sinnesfunktionen der menschlichen Haut, Abhandl. d. math. phys. Cl. d. k. sächs. Gesellsch. d. Wissensch. **23**:208, 211, 212 and 228, 1897.

99. Goldscheider: Thermorezeptoren, in Bethe, A., and others: Handbuch der normalen und pathologischen Physiologie, Berlin, Julius Springer, 1926, vol. 11, pt. 1, p. 131.

100. Blix, M.: Experimentelle Beiträge zur Lösung der Frage über die specifische Energie der Hautnerven, Ztschr. f. Biol. **20**:141, 1884.

101. Marx, E.: Die Empfindlichkeit der menschlichen Hornhaut, Leipzig, S. Hirzel, 1925.

102. Stüdemann, H.: Quantitative Prüfung verschiedener Anaesthetica in ihrer Wirkung auf die Hornhaut, Arch. f. Ophth. **115**:119, 1924.

103. Pflimlin, R.: Zur klinischen Unterscheidung verschiedener Formen des Herpes corneae, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **48**:212, 1930.

There has been a lengthy discussion about the qualities of sensation which can be perceived by the cornea. Von Frey supposed that it is possible to perceive only pain and cold, because in this membrane there are anatomically only two kinds of sense organs: the free ends of nerve fibers in the epithelium and the little bodies of Meisner-Krause. On the other hand, Goldscheider emphasized the ability of the cornea to perceive also the simple sensation of touch, if the stimulus is feeble enough. I am unable to agree with von Frey in his statement that one cannot judge the quality of sensations by perceptions but can consider it only with regard to the anatomic apparatus. Finally, perception alone has to decide the qualities of the sensations. Therefore, Stein [104] declared that the thresholds for touch and for pain in the cornea were almost equal, a finding which seems to be confirmed by the observations made in Basel, to which I have referred.

However, the hair method introduced by von Frey is a very good one and permits one to recognize the degree of the sensitiveness of the cornea in normal and in pathologic conditions much more exactly than when using a few fibers of cotton wool with an unknown stimulus value. Admittedly, the von Frey hair test has sources of error, but I shall not deal with these.

Many investigations by von Frey's method have been carried out to measure the efficiency of anesthetics on the cornea. Figure 56 shows some curves prepared by Stüdemann,[102] which demonstrates how differently some anesthetics affect the sensitiveness of the cornea. Studies were first made on rabbits. A slight winking of the lids was taken as an indication that the animal perceived the stimulus.

When the thresholds of the human cornea are tested, the patient states when he feels a sensation. By this method one can satisfactorily determine whether the sensitiveness is diminished or not, because the mean threshold in the center of the normal cornea corresponds to a hair of from 0.2 to 3 Gm. per square millimeter. The sensitiveness changes from point to point. Figure 57 shows a diagram published by Strughold [105] showing the different levels of thresholds. It will be noted that the sensitiveness is lower in the periphery of the cornea than in the center.

It appears that about 10 per cent of human beings have a higher threshold in the cornea, equivalent to a value of from 10 to 15 Gm. per square millimeter (Pflimlin [103]). These, too, are individual differences, the clinical value of which has not yet been established. It is not known, for instance, whether these corneas are more liable to

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104. Stein, H.: Welche Empfindungsqualitäten vermittelt Hornhaut und Bindehaut des menschlichen Auges? Klin. Wchnschr. **4**:819, 1925.

105. Strughold, H.: Die Sensibilität der Horn- und Bindehaut des normalen menschlichen Auges, Zentralbl. f. d. ges. Ophth. **19**:353, 1928.

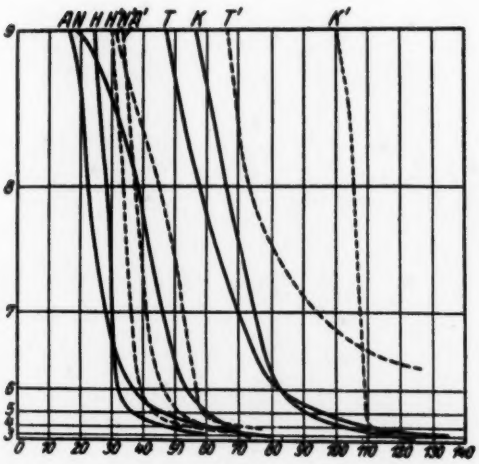

Fig. 56.—Decrease of the anesthesia after the use of various anesthetics. *K* indicates 4 per cent cocaine hydrochloride; *T*, 4 per cent tutocaine hydrochloride; *H*, 2 per cent phenacaine hydrochloride; *N*, 4 per cent procaine hydrochloride; *A*, 4 per cent alypin; *K'*, 4 per cent cocaine hydrochloride plus epinephrine hydrochloride; *T'*, 4 per cent tutocaine plus epinephrine hydrochloride; *H'*, 2 per cent phenacaine plus epinephrine hydrochloride; *N'*, 4 per cent procaine hydrochloride plus epinephrine bitartrate, and *A'*, 4 per cent alypin hydrochloride plus epinephrine bitartrate. The powers of the stimulus hairs are marked on the Y axis in tension values; the abscissas indicate time in minutes. (After Stüdemann.[102])

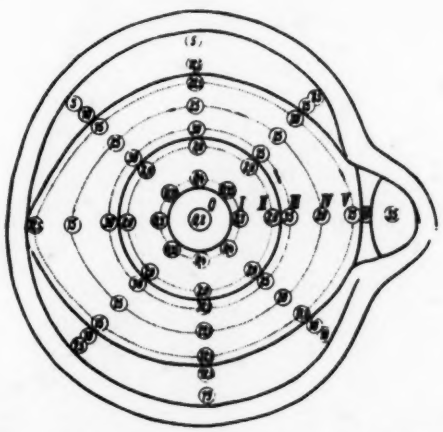

Fig. 57.—The thresholds on the cornea and conjunctiva of the human eye. The numbers indicate the pressure values of the stimulus threads (or stimulus hairs) in grams per square millimeter. (After Strughold.[105])

attack from herpes corneae, a disease which is always accompanied by a diminution of the corneal sensitivity.

The cold sensation of the cornea, studied especially by Strughold [105] has not been studied clinically. I shall merely demonstrate the distribution of the points where cold can be perceived from an illustration published by this author (fig. 58).

I believe that a more detailed investigation of the different qualities of sensation in the anterior segment of the eye may give interesting results.

DISEASES OF THE EYE DEPENDENT ON CONSTITUTION AND ON CLIMATE

I have repeatedly spoken about the individual differences in the sensory function of the eye. This difference of reaction between one person and another may be inherited or acquired.

Fig. 58.—The topography of the cold points in the anterior part of the human eye. (After Strughold, H., and Karbe, M.: Ztschr. f. Biol. **83**:2075, 1925.)

I shall consider some examples of diseases of the eye which demonstrate the variable reactions of different persons to the same influences or factors causing diseases. I shall touch on a question which is of great interest to modern medicine, namely, how the congenital constitution influences pathologic processes. The same disease never takes exactly the same course in 2 cases. The infectious diseases especially show extreme variations. Formerly, when dependence on bacteriology was at its height, these differences were attributed to the variability of the microorganisms. Nowadays, differences in the constitution of the affected person are given a much greater weight than twenty-five years ago. Undoubtedly, this brings up difficult problems, the extent of which is not fully realized and the solution of which is still far distant. Of the manifold ophthalmologic subjects of this type, I shall discuss only two: The first is tuberculous diseases of the eye, and the second, the dependence of some diseases on weather and climate.

TUBERCULOUS DISEASES OF THE EYE AND GENERAL CONSTITUTION

I shall consider the real tuberculous diseases of the eye, namely, those which are produced by a metastatic infection by the bacillus of tuberculosis itself, and of those conditions, only those of the uvea, namely, iritis, cyclitis and choroiditis. I shall also say a few words about scrofula of the eye as a tuberculotoxic condition.

It was Julius von Michel, of Würzburg, Germany, who fifty years ago recognized that many inflammatory conditions of the uvea are tuberculous. Formerly all ophthalmologists, like those in France today, considered the majority of these disorders to be syphilitic. Certainly the cause depends on the locality, but now, with modern improvements in the methods of clinical investigation and by means of the Wassermann test, the cause of the condition can be ascertained in the majority of the cases.

I shall confine myself to a discussion of research which shows that the type of reaction to disease is dependent on general constitution and age.

It is well known that the primary infection in an animal takes an entirely different course from that of a secondary infection. Similar observations can be made in cases of tuberculous conditions of the eye, with their frequent relapses. While the first attack of tuberculous iridocyclitis is usually accompanied by severe symptoms of inflammation, these are often missing in relapses. Therefore, it sometimes happens that the patients themselves do not recognize the new attack. This is an example of the different reactions of the local tissue to the poison of the tubercle bacillus. Probably a local immunity is set up. However, in some cases the first attack also proceeds quietly, owing perhaps to the different mass of the bacteria carried into the eye by the blood stream or to a preexistent local immunity.

With regard to the frequency of tuberculous conditions of the eye, some important facts have been discovered. Ophthalmologists in the northern and southern parts of central Europe noticed that to the south of the Main River and in the region of the Rhine these conditions are much more frequent than in the northern parts. For instance, in Königsberg I observed only a single case of tuberculous choroiditis during four years, while I frequently saw cases in Würzburg and in Basel. The same difference has been noted by Axenfeld between Rostock and Freiburg, Germany, and by Löhlein between Griefswald and Freiburg. These authors were completely at a loss for an explanation.

In my opinion, the influence of race is important. This suggested itself to me while I was reading a book by the psychiatrist Kret-

schmer.[106] He expressed the opinion that the genial temperament relatively frequent in the southern part of Würtemburg is the result of the mixture of the different types of the race. In the northern part of Germany tall, slender, asthenic or athletic persons are to be met with and are perhaps now better known through political considerations than from exact measurements by anthropologists. There is a remarkable bodily difference between the types called asthenic and athletic and the smaller and broader type, with the so-called pyknic construction of body, so prevalent among the inhabitants of the Alps.

The first two types, as Kretschmer[107] suggested, are correlated with the so-called schizothymic psychic behavior; the latter type, with the cyclothymic. The latter are the persons who change in their reactions and alternate between optimism and pessimism; they are extroverts, while the others are introverts. When these two physical and psychic types are mixed, as at the boundaries of the two parts of central Europe, internal psychic strained relations result, and these are often productive of genius.

To suppose that differences of bodily constitution would influence the frequency of ocular tuberculosis was obvious. The tall, slender persons of asthenic build are more frequently attacked by tuberculosis of the lungs than the small, broadly built type. Still, the general impression suggested is that these pyknic types are especially affected by ocular tuberculosis but not by progressive tuberculosis of the lungs, as I shall show.

Some measurements made in Basel by my colleague, Professor Gigon, of patients with tuberculosis of the eye suggest that this idea may be right. When compared with the measurements obtained by Neuer and Feldweg[108] in Germany for patients with tuberculosis of the lungs, certain differences are apparent. The mean circumference of the chest is different; also the weight in relation to height (tables 5 and 6). If calculations are made with the indexes of Rohrer and Bornhardt[109] (tables 7 and 8) it will be seen that these patients represent the pyknic type and are in a better general bodily condition.

In Rohrer's index the values between 1.11 and 1.31 are the limits for the asthenic type; from 1.36 to 1.49, for the athletic type, and from 1.47 to 1.58, for the pyknic type. Therefore, the majority of these patients, especially the women, correspond to the last type.

106. Kretschmer, E.: Geniale Menschen, Berlin, Julius Springer, 1929.

107. Kretschmer, E.: Körperbau und Charakter, ed. 2, Berlin, Julius Springer, 1922.

108. Neuer, I., and Feldweg, P.: Körperbau und Lungenphthise: Beitrag zur Konstitutionswissenschaft, Ztschr. f. d. ges. Anat. (Abt. 2) **13**:88, 1927.

109. Rohrer and Bornhardt, cited by von Rohden, F.: Methoden der konstitutionellen Körperbauforschung, in Abderhalden, E.: Handbuch der biologischen Arbeitsmethoden, Berlin, Urban & Schwarzenberg, 1925, vol. 9, pt. 3, chap. 4.

TABLE 5.—*Average Circumference of Chest of Patients with Tuberculosis of the Eye*

Body Height, Cm.	Number	Results in Basel Clinic, Cm.	Results of Neuer and Feldweg, Cm.
	Men		
160.6-165.5	9	88.1	87
165.6-170.5	6	92.0	88
170.6-175.5	3	86.8	89
175.6-180.5	1	99.5	90
	Women		
145.6-150.5	4	82.6	78
150.6-155.5	15	84.9	80
155.6-160.5	14	83.1	82
160.6-165.5	11	87.5	84
165.6-170.5	3	85.3	86
170.6-175.5	1	87.5	88

TABLE 6.—*Average Weight of Patients with Tuberculosis of the Eye*

Body Height, Cm.	Number	Results in Basel Clinic, Kg.	Results of Neuer and Feldweg, Kg.
	Men		
160.6-165.5	9	66.12	57.5
165.6-170.5	6	72.6	60.5
170.6-175.5	3	66.9	63.5
175.6-180.5	1	81.8	66.5
	Women		
145.6-150.5	4	58.9	44
150.6-155.5	15	64.1	48
155.6-160.5	14	63.4	52
160.6-165.5	11	68.9	56
165.6-170.5	2	66.5	60
170.6-175.5	1	85.5	64

TABLE 7.—*Measurements of Patients in Basel According to Bornhardt's Index* *

Men		Women	
	Number		Number
<0	1	<0	2
+0.1 to +3.5	3	+0.1 to +3.5	6
+3.6 to +9.0	11	+3.6 to +9.0	16
>+9.0	5	>+9.0	25

* $J = \text{weight} - \frac{\text{Circumference of Chest} \times \text{Body Height}}{240}$.

TABLE 8.—*Measurements of Patients in Basel According to Rohrer's Index* *

Men		Women	
	Number		Number
<1.33	4	<1.33	4
1.33 to 1.40	2	1.33 to 1.40	7
1.41 to 1.50	7	1.41 to 1.50	5
>1.50	7	>1.50	33

* $J = 100 \times \frac{\text{Body Weight}}{\text{Body Height}}$.

Concerning Bornhardt's index it may be remembered that + 9 corresponds to a very good state of nutrition, + 3.5 shows sufficient nutrition and a negative value indicates a bad condition.

It must be remembered that the investigations made by my colleague and myself were chiefly confined to elderly persons, while Bornhardt's index is derived from younger ones. Further, it must not be forgotten that women after the climacteric become heavier, which, however, does not prove that the general state of nutrition is really better but indicates rather that the tissues may contain more water. Nevertheless, I think that these results perhaps give a hint for the elucidation of geographic differencs in the frequency of tuberculous diseases of the eye.

A point which seems to be of importance is the preponderance of women over men. The first attack especially happens at a definite age. Concerning this point we found from a series of 100 cases of tuber-

TABLE 9.—*Age of Onset of Tuberculosis of the Eye in Males and Females (After Achermann*)*

Age	0-9 Yr.	10-19 Yr.	20-29 Yr.	30-39 Yr.	40-49 Yr.	50-59 Yr.	60-69 Yr.	70-79 Yr.	Total
Female	..	10	9	15	18	14	3	1	70
Male	1	10	10	2	6	1	..	..	30
Sum	1	20	19	17	24	15	3	1	100

* Achermann, E.: Klinisch-statistischer Beitrag zur Kenntnis der Iridocyclitis tuberculosa, Schweiz. med. Wchnschr. 56 : 1119, 1926.

culosis of the eye that the beginning of the illness occurs in the majority of patients during the fifth or sixth decade (table 9). This holds good also when considered in relation to population. The second maximum of tuberculous disease, during the second and third decades, is apparent only if the figures are considered in relation to the whole population.

Consideration of sex incidence shows that the disease is more frequent in women than in men (table 9) in the fifth decade. Perhaps there is a second peak in the second decade, but the number of cases is not sufficient to justify a definite conclusion. In the male the peak lies in the second and third decades; the minimum in the fourth is not certain, owing to the small number of cases.

These differences in the onset of tuberculosis of the eye are certainly due in women to a diminution of the power of resistance during the climacteric and perhaps also to similar changes at the beginning of sexual maturity. The maximum for the males in the second and third decades may be caused by increased demands of the daily work which the youthful organism is not yet fully able to meet.

The view that iridocyclitis in elderly women is mostly of tuberculous origin, and not of a completely unknown causation, is nowadays, I believe, generally adopted in Central Europe, although the Austrian ophthalmologists for a long time would not accept it.

All tuberculous conditions of the inner parts of the eye are metastatic, arising from some other focus in the body. This focus in nearly all cases lies in the lungs or in the glands of the chest. Accordingly, the first intraocular attack often follows an attack of influenza or grip, by which an incompletely healed focus becomes mobilized and the bacteria reach the eye or some other organ. This stage of the illness corresponds to that of generalization and coincides with the second stage of Ranke.[110] It lasts many years.

Although it must be assumed that in all cases of intraocular tuberculosis there is an older focus in the chest which is still active or which has been reactivated, many authors have emphasized that even in cases of serious involvement of the eye disease cannot be found clinically in the lungs. There is especially no progressive focus in the lungs, and the patients seldom die in consequence of tuberculosis of the lungs. Some authors formerly suggested that when nothing is to be found in the lungs, the cause of iridocyclitis is probably tuberculosis! This opinion is certainly not justified, because when one examines the patients with roentgen rays some tuberculous lesions of the chest are usually found. It is certainly often impossible to say whether or not there is an active process in the lungs.

But Werdenberg,[111] of Davos, Switzerland, emphasized that if a thorough general examination is made evidence of tuberculosis is likely to be found in the chest. He found that in 60 per cent of more than 1,000 cases the condition in the chest was insignificant, but in 30 per cent it was serious. However, it must be remembered that the patients sent to Werdenberg in his sanatorium in Davos usually have a serious or extreme condition.

A correlation between the form of the disease in the chest and that in the eye could not be established. The majority of relapses are certainly caused by incompletely cured conditions in the chest.

Intraocular tuberculosis is, as I have shown, to be compared to the other metastatic forms in the body. Rollier[112] at Leysin, demonstrated that general surgical tuberculosis also is not associated with severe lesions in the lungs. Perhaps it is of interest that my assistant

110. Ranke, K. E.: Primäraffekt, sekundäre, und tertiäre Stadien der Lungentuberkulose, Deutsches Arch. f. klin. Med. **119**:201, 1916.

111. Werdenberg, E.: Zur Beurteilung und Behandlung der Augentuberkulose, Klin. Monatsbl. f. Augenh. (supp.) **94**:1, 1935.

112. Rollier, A.: Die Heliotherapie der Tuberkulose mit besonderer Berücksichtigung ihrer chirurgischen Formen, ed. 2, Berlin, Julius Springer, 1924.

Dr. Achermann during his investigation of 100 cases of tuberculosis of the lungs at Leysin several years ago found only 1 patient with ocular tuberculosis.

The explanation of this difference between the state of the eye and that of the lungs is at present far from clear. There must be some special reason for this immunity, which at present is not known but which certainly depends, as already observed, on the general constitution of the body. Nevertheless, intraocular tuberculosis must always be treated not only by local medication but by general measures. A stay in the mountains for many months often brings about surprising results. I have seen this in many cases. The former, and even present day, use of tuberculin without definite indication should be avoided.

The importance of early complete healing of the local process in the eye is seen in table 10 (Achermann). It shows the correlation between the duration of the ocular disease and the diminution of the

TABLE 10.—*Decrease of Visual Acuity with Increasing Duration of Tuberculous Disease of the Eye (After Achermann)*

Visual Acuity												
6/5	4	3	3	—	—	—	—	—	—	—	—	—
6/6	15	6	2	1	—	—	—	—	—	2(1)	—	—
6/8	10	2	2	1	—	—	—	1	—	(1)	—	—
6/12	9	5(1)	—	2	1	—	1	—	—	1	—	—
6/18	7	4(1)	2	1	1	—	—	—	—	2(1)	1	1
6/24	2(1)	—	2	1	—	—	—	1	—	1	1	1
6/36	1	—	—	—	—	—	1	—	—	1	—	1
6/60	1	3(2)	1	3	—	—	1	1	—	2	1	1
Counting fingers at 1-2 m	—	3(1)	3	4	—	4	2	—	—	4	3	7
Light sensation	—	—	—	1	4	1	1	3	2	1	1	7
Duration of the disease, yr	1	2	3	4	5	6	7	8	9	10	15	20

visual acuity in the cases in which relapses occurred. Good visual acuity is to be found chiefly in cases of involvement of less than four years' duration. The visual acuity is seen to decrease as the duration of the illness increases. This is due to the exudates formed by the inflammation and later often to complicated cataract. The table proves that by every means available complete cure of the condition in the eye must be attempted at its commencement.

Scrofula of the Eye.—When one considers the relation between the local tuberculous condition in the eye and the primary focus in the chest, a certain degree of antagonism is found. In contrast to this fact are the conditions which are to be found in the cases of scrofula. This illness is also one involving the whole body. It manifests itself principally on the skin and on the surface of the eye (the conjunctiva and cornea).

Scrofula is at present regarded as a tuberculotoxic condition with a hypersensitiveness of the integument, caused by the toxins of the tubercle bacillus. Children especially are affected, apparently in con-

nection with a primary infection in the lungs. Therefore, the typical phenomenon of scrofula of the eye, the phlyctenule, is not due to the tubercle bacillus but is to be regarded as a specific reaction to the toxin. Admittedly the endogenous factor is not the only one, for exogenous factors, such as uncleanliness, bodily strain and lack of fresh air, can all play their part in producing the condition.

In scrofula of the eye, a closer connection exists between the severity of the local focus in the eye and that in the chest than in true tuberculosis. Table 11, published by Stalder,[113] shows the results in 100 cases of scrofula of the eye in which the state in the lungs was determined by roentgen examination in the General Policlinic in Basel. In only 17 cases were there negative or uncertain findings in the chest. The examinations were made with the fluoroscope for reasons of economy,

TABLE 11.—*Intrathoracic Condition in Patients with Scrofulous Inflammation of the Eye (After Stalder)*

Age in Years	Negative or Uncertain Diagnosis	Hilus		Condition of Lungs Without Physical Confirmation	Condition of Lungs with Physical Confirmation	Total Number	
		Condition of Hilus	With Calcification			Positive	Altogether
0- 5	3	7	1	1	8	16	19
6-10	3	10	5	2	7	19	22
11-15	2	11	6	3	7	21	23
16-20	5	3	2	4	1	8	13
21-25	1	2	2	4	3	9	10
Over 25	3	0	3*	3	4	10	13
	17	33	19	17	30	83 : 100	

* Without enlargement of hilus.

but the same results would have been obtained had photographs been made.[114]

The patients with severe scrofulous conditions of the eyes are often found to have marked alterations in the lungs. A typical enlargement of the hilus was observed in children under 5 years of age. In patients more than 5 years old this was combined with calcified foci or was absent, indicating a tendency toward healing. After the sixteenth year the number of paitents having lesions of the lungs increases. In the third decade and later the number of positive findings in the lungs diminishes.

Generally, one may say that scrofula of the eye is always associated with alterations in the chest of no small degree but apparently of no great malignancy. Therefore, some physicians have recommended mar-

113. Stalder, H.: Zur Kenntnis der Lungenbefunde bei Augenskrophulose, Schweiz. med. Wchnschr. **56**:618, 1926.

114. It may be mentioned that the dark-adapted eye when looking at the fluorescent screen has a very low visual acuity. When the light-adapted eye looks at photographs it can distinguish considerably more detail.

riage with women who had had scrofula in their childhood, because of the protection which this gives in later life against progressive tuberculosis of the lungs. This seems to be only partly justified.

DISEASES OF THE EYE IN RELATION TO WEATHER AND CLIMATE

The significance of climate or of definite influence of the weather on the incidence of some general diseases has been noted occasionally. But in recent years the interest in this problem has increased.

Among others, Hellpach,[115] in Germany, studied the influence of the weather on man. In Switzerland, Fritzsche [116] affirmed that under the influence of the foehn wind the frequency of apopleptic seizures, embolisms and thromboses increased. At Innsbruck, in Austria, for instance, where the foehn wind is very strong, the physical and mental energy of those who are sensitive to this phenomenon of the atmosphere is considerably diminished. Surgeons do not operate during this time unless it is absolutely necessary, because of the decreased resistance both of the patient and of the physician.

The typical foehn is a so-called falling wind (*Fallwind*), which approaches the heights as a warm wind from the south. On the ridge of the mountains it becomes colder and therefore heavier. It then descends rapidly and in doing so again becomes warmer. Violent storms, accompanied by manifold disturbances in the atmosphere are frequent in consequence.

The precise way in which the foehn affects the human body is still unknown. No opinions in regard to this problem have been confirmed.

The factors which change with the weather and which are ordinarily registered by the meteorologic stations are: temperature, intensity and direction of the wind, clouds, precipitation and atmospheric pressure. Recently the degree of cooling (*Abkühlungsgrösse*) also has been recorded by some institutes, for instance, in Basel and Davos.

The degree of cooling takes account not only of the temperature but of all factors which cause loss of warmth to a small black sphere of metal which is heated electrically to 36.5 C. The sphere is placed, for instance, on the roof of a house. The amount of electric current necessary to keep the black sphere at a constant temperature of 36.5 C. is then measured.

Cooling depends on several factors: the temperature, the wind, the rate of emission of heat from the black sphere into the surrounding air and the absorption by it of heat from the sun. It is clear that the same factors which are of importance in such an example of cooling influence the living organism in a similar manner. Therefore, this index may be expected to be of greater biologic interest than that of temperature or of the wind alone.

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115. Hellpach, W.: Geopsychische Erscheinungen, ed. 3, Leipzig, Wilhelm Engelmann, 1923.

116. Fritzsche, E.: Witterung—Thrombose und Lungenembolie, Schweiz. med. Wchnschr. **60**:889 and 1055, 1930.

Some authors have suggested that the free ions in the atmosphere also influence the human body, especially the vessels. But determinations made in the meteorologic laboratory at Davos, Switzerland, under the direction of Mörikofer [117] proved that the number of positive and negative ions, both large and small, changes rapidly in a room, for instance, without any influence on the human body. Therefore, this factor is probably not of importance.

I recently investigated the correlation between the state of the weather and the incidence of certain diseases in the ophthalmologic clinic in Basel. I obtained some interesting facts. The observations extend over a period of nine years, from 1926 to 1934.

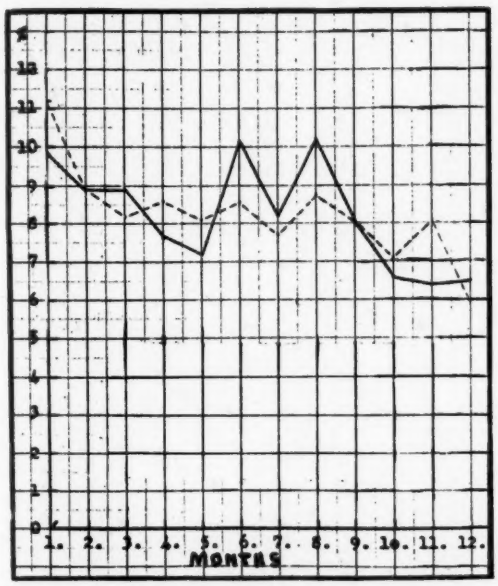

Fig. 59.—Hordeolum from 1926 to 1934. Frequency in percentage for a month of thirty days. The dash line indicates the frequency curve for the policlinic.

Hordeolum.—I shall refer briefly to hordeolum. I have not distinguished here between disease of the large meibomian glands and that of the small Zeiss glands in the outer surface of the lid at the seat of the eyelashes. These glands have almost the same function and are also generally exposed to the same noxious conditions.

In regard to the fluctuation in the frequency of this disease in relation to the seasons, figure 59 shows that there are three periods of incidence, namely, in January, June and August. A minimal number of cases are to be found in May and in the last three months of the year.

117. Mörikofer, W., and Chorus: Ergebnisse con Ionenzählungen in Davos, Verhandl. d. Schweiz. naturforsch. Gesellsch., 1932, p. 324.

That this increased frequency is not dependent on the number of patients attending the policlinic at Basel is easily seen from the diagram in figure 59. Therefore, the variation in frequency of this disease must be significant, all the more so as the number of cases (about 1,000) seems sufficient for conclusions to be drawn.

On correlation of the factors concerning the weather with the onset of hordeolum in each case, an undoubted dependence on temperature was found, especially as regards fall in temperature or a high degree of cooling. Figures for the degree of cooling are available in Basel only since 1929. Table 12 shows that in two thirds of all cases of hordeolum the condition is related in onset to a fall in temperature and an increase in cooling rate. The fall in temperature occurred one or two, seldom three, days before the appearance of the sty. This

TABLE 12.—*Hordeolum; Dependence on Lowering of Temperature and Increase in Degree of Cooling*

	Temperature			Cooling		
	+	−	Total	+	−	Total
1929	62	34	96	65	31	96
1930	88	47	135	99	36	135
1931	81	45	126	94	32	126
1932	82	44	126	82	44	126
1933	91	43	134	83	51	134
1934	81	45	126	84	42	126
Total	485	258	743	507	236	743

Increase of degree of cooling 1929-1934	507	:	236	=	68.2%	:	31.8%
Decrease of temperature 1929-1934	485	:	258	=	63.9%	:	34.7%
Decrease of temperature 1926-1934	698	:	347	=	66.9%	:	33.2%
	(1,045)						

time, as is to be expected, corresponds to the period of incubation. Considering the large number of cases, coincidence can be ruled out. In my opinion, the skin of the eyelids, being exposed to the influence of weather, is cooled and consequently has a lowered resistance to the bacteria, especially staphylococci, which are present in the skin. The lowering of the resistance of the tissues permits the onset of inflammation. This explanation accords with the opinion, often expressed by the patient, that he had been chilled before the hordeolum appeared.

It is to be noticed that long periods of cold do not increase the number of cases. It is only in the beginning of the period that cases become more frequent, especially when the cold sets in after a period of warm weather.

It seems to me that this fact proves that the skin and its organs were previously adapted to a higher temperature and are not able to accommodate themselves quickly enough to the lower temperature.

But there remain about one third of all cases which cannot be explained in this way. Here other influences may be at work. It is

especially noticeable that during the summer an extremely high temperature frequently coincides with an outbreak of hordeolum. This may be due to the patients' having exposed themselves to a higher degree of cooling, say in a draught, with the intention of seeking relief from the heat. The onset of the hordeolum may be explained in the same way as before. But it seems more probable that copious perspiration of the face leads to a rubbing or touching of the lids with the fingers or pocket handkerchief and a consequent infection of the glands. This suggestion would be in accord with the fact that many persons have general inflammation of the follicles in the skin after perspiration.

Catarrhal Ulcer of the Cornea.—Conditions similar to those which favor the occurrence of hordeolum also accompany the onset of the so-called marginal or catarrhal ulcer of the cornea. This small ulcer, which lies from 0.5 to 1 mm. from the margin of the cornea, is benign but tends to relapse for some years afterward. It occurs mostly in middle-aged or elderly persons. The relapses suggest that some constitutional factor may be at work.

It was noticed that often during the summer when a few cold days occurred suddenly this disease was fairly frequently encountered. The statistical treatment of about 300 cases confirms these observations, because the highest frequency is to be found from July to October, while during the winter it is relatively low. There are two minima: one during May and June, and the second in November and December (fig. 60).

Consideration of the meteorologic factors has made it possible to establish here too a definite connection between a fall in temperature or an increase in the degree of cooling and the onset of the ulcer. There is therefore a strict positive correlation between the beginning of this ulcer and the state of the weather. In about three fourths of the cases there is such a connection (table 13).

This relation is especially clear in the summer months. It may be explained by the absence of vessels in the cornea and the lower temperature of that membrane in relation to the rest of the body. When the cornea during the warm season has been adapted to a high temperature, cool weather causes an exaggerated loss of warmth and thereby diminishes its resistance. Since there are no specific bacteria which cause the ulcer, a constitutional factor must certainly be responsible, of what nature it is difficult to say. Perhaps defective adaptation of the cornea to extreme temperatures is due to an insufficient regulation of the sensitiveness of the vessels in the immediate neighborhood of the cornea. It would be of interest to determine whether these patients show any similar alteration of the general vessels. The method of Otfried Müller, the so-called microscopy of the capillaries, might be useful for this purpose.

Acute Conjunctivitis.—In contrast to these ocular diseases, investigations concerning acute conjunctivitis gave other results. This disease is certainly in some cases due to local cooling; it is, for instance, often observed after a person has driven a motorcar with open windows, so that a draught of air struck the eye. Since in cases of acute conjunctivitis pneumococci are usually found, it is justifiable to suggest that the damage to the anterior part of the eye facilitated attack by the bacteria through diminishing the defensive power of the organ.

But examination of the cases of acute conjunctivitis for nine years shows no strict dependence on the temperature, although there is a

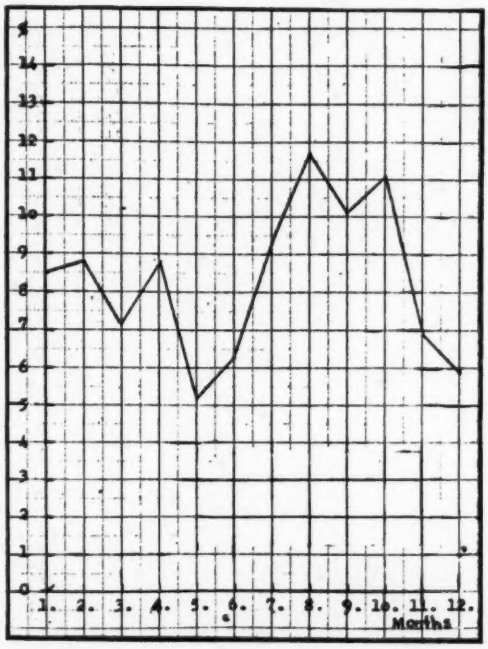

Fig. 60.—Ulcus catarrhale from 1926 to 1934. Monthly frequency in percentage.

TABLE 13.—*Ulcus Catarrhale; Dependence on Lowering of Temperature and Increase in Degree of Cooling*

	Temperature			Cooling		
	+	−	Total	+	−	Total
1929	30	15	45	33	12	45
1930	25	6	31	23	8	31
1931	34	2	36	27	9	36
1932	27	6	33	21	12	33
1933	28	4	32	28	4	32
1934	27	11	38	30	8	38
Total	171	44	215	162	53	215

Increase of degree of cooling 1929-1934	162 : 53	=	75.3%	: 24.7%
Decrease of temperature 1929-1934	171 : 44	=	79.5%	: 20.5%
Decrease of temperature 1926-1934	238 : 68	=	77.9%	: 22.2%
	(306)			

summit of incidence in May and another in October. The latter could be taken as produced by a fall of temperature. But closer consideration does not support this suggestion. On the contrary, about a half or two thirds of the cases have no relation to the temperature or the degree of cooling. I think it must be concluded that the onset of acute conjunctivitis is determined by different factors, partly bacteriologic and partly of other kinds. It may be especially stated that the cases mentioned do not comprise instances of severe infections, such as diphtheria, gonorrhea and inflammation with the so-called inclusion bodies (*Einschlusskörperchen*), namely, trachoma and swimming pool conjunctivitis.

This negative result seems to me to prove that the dependence of the two previously mentioned diseases on the weather is real.

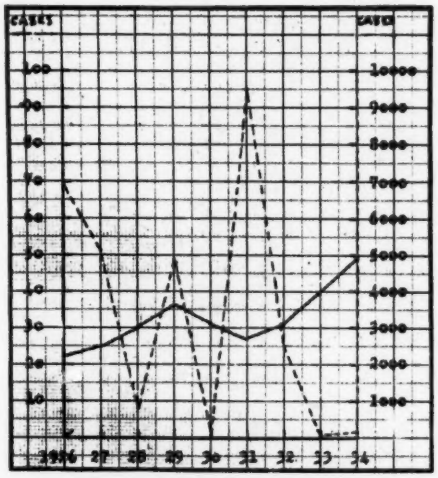

Fig. 61.—Relation of the frequency of herpes corneae to the frequency of influenza. The continuous line indicates cases of herpes; the dotted line, cases of influenza.

Herpes Corneae.—Another ocular disease which probably depends on the weather is herpes corneae, the eruption of small vesicles on the surface of the cornea, which frequently is followed by an extension to the deeper parts of the membrane (keratitis disciformis). Although it was learned from the experiments of Grüter and Dörr that the condition must be caused by an infective ultramicroscopic virus, it is conceivable that the weather may influence the frequency, for instance, by increasing the disposition to the disease. However, a more detailed investigation demonstrated no relation to the temperature in general. Certainly in some cases a cold may be the cause.

The statement in textbooks that herpes corneae follows influenza hardly seems to be justified by statistics. Figure 61 shows the frequency

of the cases of herpes observed in the clinic in Basel and the number of cases of influenza reported to the sanitary department in Basel during a period of nine years. There is no correlation. This proves, as some authors have believed, that the causes of influenza and herpes corneae, at present unknown, are not identical. However, there may be some relation.

Hinrichs,[118] of Greifswald, after investigations extending over a period of only two years expressed the opinion that the weather, especially "the fronts," has an influence on the appearance of herpes. The results of his investigation do not seem to me to be sufficiently conclusive.

Scrofula of the Eye.—Of more interest is the relation of scrofula of the eye to the seasons. It has been seen that this disease is due to a tuberculous infection and that the manifestations on the skin and in the eye are not really tuberculous, but only toxic. The incidence of this

TABLE 14.—*Scrofulous Conjunctival and Corneal Conditions*

Years	Number of Cases	Total Number of Outdoor Patients
1926	191	4,754
1927	155	4,837
1928	193	5,225
1929	254	5,589
1930	192	5,642
1931	190	6,000
1932	195	7,061
1933	261	6,670
1934	151	6,796
Total	1,782	52,574

disease has greatly diminished in all countries of middle Europe during the last fifteen years, perhaps in connection with the improved hygienic conditions of the dwellings and with better nourishment. Nevertheless, in Basel a change in its frequency has been observed during recent years which is by no means explained (table 14). A relation to the change of the weather from year to year is not established. But the dependence on the season is remarkable (fig. 62). An increase of the cases in the first half of the year, especially from March to June, is followed by a decrease during the last months of the year. It is due not only to a decrease in the number of patients in general, but apparently to the influence of the seasons, which effects a change in the resistance of the body.

Nearly the same course of the curve was to be found in Palermo, Graz, Greifswald and Helsingfors. The summit varies according to

118. Hinrichs, H.: Einfluss von Wetter und Jahreszeit auf die Entstehung von Augenkrankheiten, Ztschr. f. Augenh. **86**:269, 1935.

the geographic situation of the town, whether it is in the south or in the north. There must be a common cause which affects the frequency, dependent on the seasons (Rohrschneider [119]). A similar distribution during the different seasons was also found by Essen-Möller in Lund.[120]

I suggest that the cause is to be found in the favorable effect of the air and sun in summer on the tuberculous process in the chest. The diminution of the tuberculous toxins produced reduces the frequency of scrofulous manifestations. On the contrary, the dark winter, with the small amount of sunshine and confinement to the house, diminishes the resistance. These influences last until the late spring. Rohr-

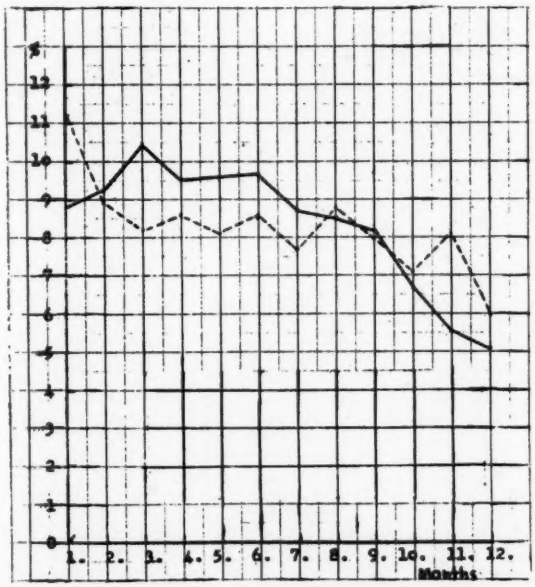

Fig. 62.—The continuous line indicates scrofula from 1926 to 1934 (frequency in percentage for a month of thirty days). The dash line indicates the frequency curve for the policlinic.

schneider [119] expressed the same opinion. One might believe that the increasing insolation in the spring excites the efflorescence of the scrofula in the eye. Occasionally after strong doses of artificial sunlight (the mercury lamp) an increase of recent scrofulous phenomena is seen in the eye. Perhaps both causes are effective. In considering the

119. Rohrschneider, W.: Ueber den Frühjahrsgipfel der skrofulösen Augenerkrankungen, Ztschr. f. Augenh. **86**:281, 1935.

120. Essen-Möller, L.: Eine Studie über die Häufigkeit der Phlyctaenen, Acta ophth. **14**:414, 1936.

decrease of the resistance during the winter, account must also be taken of the lack of fresh vegetables and the consequent lack of vitamins.

These questions are still theoretically unsolved, but the practical consequences are simple. If the relapses so frequent in this disease are to be avoided, scrofulous patients should go to the high mountains during the second half of the winter; thus they will escape the mist and the fog in the plains. If the financial situation does not allow this, a thorough treatment by artificial sunlight is to be recommended. Indeed, after this treatment I have observed good results even in serious involvement, especially in the absence of relapses.

Acute Glaucoma.—Finally, of great interest is the relation of attacks of acute glaucoma to the weather. In the beginning of this century Steindorff [121] called attention to the fact that such attacks were more frequent in the winter than in the summer. He stated that this was due to the cooling of the body, which produces a disturbance in the regulation of the flow of the blood. Other authors said that the darkness in winter dilates the pupil, which increases the chances of an attack. There is also the fact that economic cares, which are greater in winter than in summer, increase the disposition to an acute attack from alterations in the blood vessels.

At present there is no certain knowledge about the mechanism of acute glaucoma. That the season has great influence on the frequency is shown in figure 63. It clearly demonstrates that from November to February this disease is much more frequent than in the summer.

The frequency changes also with the years in general (fig. 64). Especially in the last few years the number of cases of acute glaucoma observed in Basel has increased. Whether social factors caused by the economic crisis operate here is still uncertain.

De Rudder [122] emphasized some years ago that acute glaucoma occurs especially at the passing of the so-called cool and warm fronts. Modern meteorology teaches that the antagonism of the warm tropical and the cold polar air is of the greatest importance to the general type of the weather. These bodies of air (*Luftkörper*) are, it seems to me, of interest to medical science also. De Rudder suggested that no special factor accompanying the change of the body of air is effective, but all factors combined. Especially it is not the pressure of the air which has an influence in producing disease.

Dr. Bider, of the Meteorological Institute in Basel, helped me to evaluate cases of acute glaucoma with regard to the weather. In 150 cases we were able to fix the time of the beginning of the attack exactly. As shown in table 15, in 87 cases the onset coincided with the passage

121. Steindorff, K.: Ueber den Einfluss von Temperatur und Jahreszeit auf den Ausbruch des akuten primären Glaukomanfalles, Deutsche med. Wchnschr. **28**: 924, 1902.

122. de Rudder, B.: Wetter und Jahreszeit als Krankheitsfaktoren: Grundriss einer Meteoropathologie des Menschen, Berlin, Julius Springer, 1931.

of a warm or cold front. Only 17 cases were observed with foehn and 19 with severe cold, and only 11 of 150 cases occurred without any remarkable disturbance in the atmosphere.

The weather is certainly to be considered as influencing the flow of blood by producing decompensation. Acute glaucoma is in my opinion

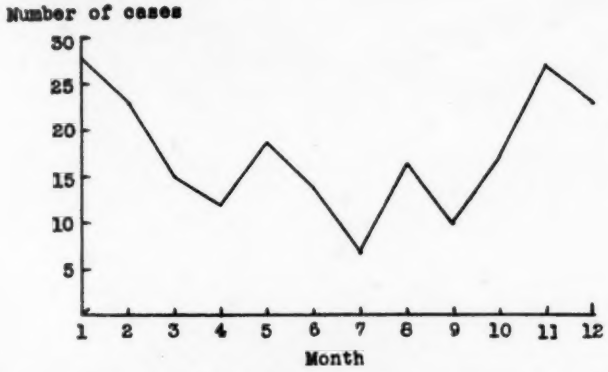

Fig. 63.—Acute attacks of glaucoma. Curve of the totals for the months compiled for the years 1920 to 1934.

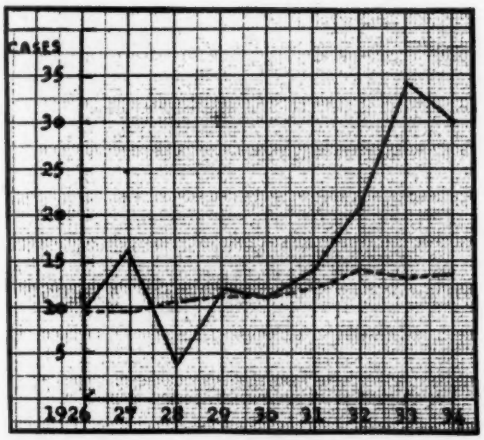

Fig. 64.—Glaucoma. Curve of the totals for the years 1926 to 1934. The dash line indicates the frequency curve for the policlinic.

related to an insufficiency of the vessels, although in what manner is not known.

It may be of interest in this connection to note the following observations made on 6 patients attacked by different illnesses at the same time, from Oct. 26 to 28, 1935, during the passage of an extreme warm front. All these illnesses, ophthalmic migraine, apoplexy, extrasystoles, and so on, result from disturbances of the vessels, which must have a common cause.

On October 26 the air on the continent was cold, while warm air was to be found above the ocean. On October 27 a definite warm front passed across the continent and reached Basel in the evening; a storm from the northwest arose there, accompanied by very warm air. Next day the front disappeared and heavy rain fell.

It is not known what factor associated with the movement of the air bodies is effective. I have recently begun investigations with a physicist and a physiologist to try to solve this complicated and important problem. Nevertheless, some practical conclusions may be deduced from the facts already known. All patients suffering from simple

TABLE 15.—*Acute Attacks of Glaucoma in the Years 1926 to 1936*

Meterorologic Condition	Number of Attacks
Cold front	42
Warm front	45
Foehn	17
Cold without front	19
Snow	2
Other disturbances	14
No disturbances	11
Total	150

TABLE 16.—*Influences of a Sudden Approach of a Warm Front on the Vasomotor System During the Period from Oct. 26 to 28, 1935*

1. Attack of acute glaucoma in an elderly man during the night of October 27; intense pains at 3 o'clock in the morning.
2. Woman of 75; several years ago acute attacks of glaucoma, at present regularly taking pilocarpine; on October 27 typical headache on the right side, passing without attack; apparently a slight increase of the intraocular pressure.
3. Physician, hemicranial migraine from October 27 to 28.
4. Physician, suffering from habitual asthma; during night of October 27 serious attack of asthma.
5. Elderly physician; on October 27, extrasystoles and irregularities of pulsation.
6. Woman nearly 70; on October 27 sudden diminution of the vision, occasioned by thrombosis in a branch of the retinal vessels.

glaucoma should, in order to minimize the danger of an acute attack, use more pilocarpine when the weather changes, especially when a "front" approaches. This can readily be ascertained from the meteorologic data published daily by the newspapers.

In general, it may be emphasized that this sensitiveness to the weather or to several meteorologic factors, which have been established for glaucoma, is caused by an individual insufficiency in the regulation of some parts or functions of the body. For those who are not sensitive, the weather does not matter. The sensitiveness perhaps changes with increasing years. I know many inhabitants of Basel, which in this respect has not a good climate, who have become more and more sensitive to the weather. Probably the faculty of adaptation of the blood vessels diminishes with their increasing rigidity.

SUMMARY

As in all other respects, so also from the special standpoint of the ophthalmologist, different persons show different reactions to normal and abnormal stimuli. Adaptation of the living substance is possible within a wide range. In relation to adequate stimuli, the visual organ is of an individual variability which is sometimes so extreme, for instance in cases of congenital color blindness, that a mutual understanding between abnormal and normal persons is impossible.

Adaptation to low intensities of light also shows considerable individual variability, so that persons with extreme degrees of insufficiency can exist only by the protection which human civilization affords.

In his environment, in a narrow sense, the individual changes by adapting himself to the given circumstances, but there are remarkable differences in reaction to some definite meteorologic changes. Human civilization allows man to protect himself against the inclemency of the weather, but this defense is only relative. There are some factors against which the power of adaptation of many persons is insufficient.

Also in tuberculous infection of the eye, which I have mentioned, there are a variety of reactions according to the hereditary or acquired constitution of the individual which led, in spite of the ordinarily equally infective virus, to different courses of the illness.

The human organism and one of its organs, the eye, react in a most variable manner. This fact teaches how surprisingly variable human life is, even in this small province.

For the purpose of classification, be it in a scientific, social or political sense, this variation in life may be disturbing or inconvenient. Therefore, it has often been overlooked or intentionally neglected. But each organism is and always will be an individual.

SIMPLE DACRYOCYSTORHINOSTOMY

LOREN GUY, M.D.
NEW YORK

The birth of the idea of the dacryocystorhinostomy occurred when the lacrimal sac and ducts were shown to be conducting structures and not secreting organs. Antoine Maître-Jan [1] appears to have been the first to have recognized the true anatomic structure and physiologic function of the lacrimal duct and sac. His work formed the foundation of rational therapy of the part. Before his time it was believed that these structures were glands.

The first surgeon to take advantage of the basic physiologic and anatomic knowledge of the lacrimal structures was the Englishman Woolhouse. Woolhouse himself left no known description of his method of operating, but his technic is mentioned by Platner [2] in an article published early in the eighteenth century. Metal cannulas and frequent irrigations were employed in the earlier technic.

Modern work began with the contributions of the Florentine rhinologist Toti,[3] in 1904. All subsequent work has been along the lines set down by Toti. Following him, the work has consisted mostly in individual preferences in method and in the introduction of special instruments.[4]

West [5] and Halle [5a] have devised an intranasal operation which appears well suited to one trained in intranasal work. It has the advantage of eliminating the possibility of a visible scar.

From the New York Eye and Ear Infirmary.

Read at the New York Academy of Medicine, Section of Ophthalmology, Jan. 18, 1937.

1. Maître-Jan, A.: Traité des maladies de l'oeil et des remèdes propres pour leur guérison, Troyes, Jacques le Febure, 1707.

2. Platner, J. Z.: De fistula lacrymali; respondente, Henrice Godofredi Heyland, Lipsiae, lit. I. Titii, 1724.

3. Toti, A.: La dacriocistorinostomia come cura radicale conservatrice delle suppurazioni gravi e delle fistole del sacco lacrimale: Resultati ottenuti nei primi sette casi operati, Policlinico (sez. chir.) **11**:545, 1904.

4. Weeks, W. W.: The Technic of Dacryocystorhinostomy, Arch. Ophth. **7**:443 (March) 1932. Dupuy-Dutemps and Bourguet: Procédé plastique de dacryocysto-rhinostomie et ses résultats, Ann. d'ocul. **158**:241, 1921.

5. West, J. M.: The Clinical Results of the Intranasal Tear Sac Operation, Tr. Sect. Ophth., A. M. A., 1931, p. 69.

5a. Halle, M.: Intranasale Tränensackoperation bei Säuglingen, Med. Klin. **24**:1699, 1928.

With the objective of developing a method of doing a dacryocystorhinostomy that would be effective and yet simple, work was started on cadavers four years ago. Three years ago the operation was applied to living subjects. A small group of patients was carefully studied and such changes were made in the method as the results indicated. In doing this work, Dr. Francis Shine, Dr. Clyde McDannald and Dr. Hugh Blackwell have been most helpful.

It has been concluded that the operation is indicated in cases of chronic dacryocystitis in adults. So many children respond to nonsurgical treatment that it should be deferred, if possible. Owing to its effectiveness in eliminating epiphora and infection in upward of 85 per cent of cases, dacryocystorhinostomy is preferable to extirpation of the sac. In cases in which the operation has not been a success, no more serious damage has been done other than the inconvenience of a minor local operation.

The operation should not be done in cases in which an intraocular operation is likely to be necessary. The dangers of an ascending infection from the nasal cavity appear too great. Obstruction in the canaliculi and in the nasal passages might render the operation useless. These structures should be examined before the operation is undertaken, and any polyps, sinus disease or anatomic malformations should be corrected. The operation should not be done in the presence of acute infection.

The technic as finally accepted is as follows:

Anesthesia is obtained by infiltrating the area with a 2 per cent solution of procaine hydrochloride, 8 parts, and epinephrine hydrochloride in a dilution of 1:1,000, 2 parts. Intranasal application of cocaine hydrochloride was found not to be necessary. Depending on the temperament of the patient, a preoperative sedative is given, usually ⅛ grain (0.008 Gm.) of morphine sulfate.

Before the incision in the skin is made, the angular vessels are located by palpation, and by avoiding them much troublesome bleeding is eliminated. The incision, *A* in the illustration, is made from a point 3 mm. above the inner canthal ligament downward and outward just nasal to the anterior lacrimal crest. The length depends on the size of the sac and the anatomy of the parts, but averages 20 mm. The incision is made down to the bone through the periorbita. The periorbita is then elevated temporally, exposing the lacrimal fossa and the sac. In the lower half of the fossa a window about 4 by 6 mm. is made. A chisel, a pair of scissors or a curet is used to make this window. Such an instrument allows for anatomic variation and seems to be more desirable than a fixed trephine or other similar instrument.

The lower half of the fossa is emphasized because there ethmoid cells are rarely, if ever, found, according to Whitnall. While it does not appear to make much difference whether or not these cells are entered, it would seem best not to disturb them.

After the window is cut through the bone of the fossa, the corresponding mucous membrane is excised, and then the corresponding portion of the sac is also excised. The tissues are allowed to fall back into their natural positions,

the excised portions coming in apposition, thus forming a new line of communication from the conjunctival sac to the middle meatus of the nose. The tissues are held in place by a pressure bandage. No sutures are used except for the skin. Fine interrupted sutures are taken there. The insertion of sutures to hold the mucous membrane of the nose and sac in place takes considerable time and, in addition, such sutures do not seem necessary. It is doubtful if the introduction of a foreign material, such as suture material, in inflamed tissues, such as chronic dacryocystitis presents, is desirable.

On the second day after the operation the pressure bandage is replaced by a dressing of collodion. The skin sutures are removed on the fourth or fifth day. It requires less than thirty minutes to perform the average operation.

In the series of cases in which this operation was performed no complications have thus far been encountered. In the literature severe

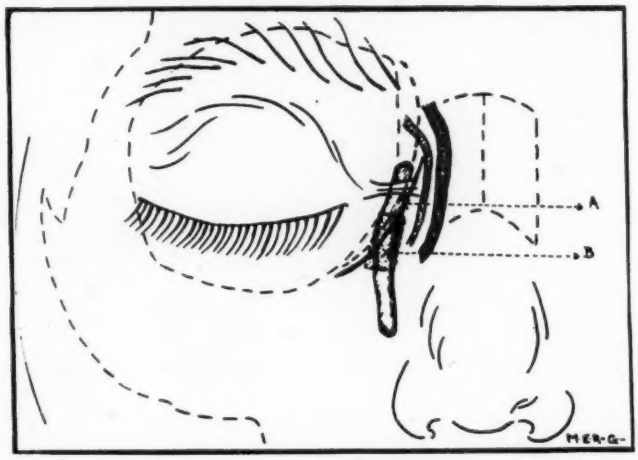

Diagram illustrating the simple dacryocystorhinostomy operation. *A* indicates the incision and *B*, the window cut in the lower half of the lacrimal fossa. Note the relation of the angular vessels.

hemorrhage from severing the nasociliary artery has been reported. For this reason it might be desirable to be prepared for such an emergency. Hematoma of the orbit and infection have also been reported, occurring with about the same frequency as with extirpation of the sac.

To date, 54 patients have been operated on by seven different operators. Only one of the operators had had previous experience with any type of dacryocystorhinostomy. Fifty of the patients were relieved of both epiphora and empyema of the sac. One failure was due to faulty technic, and one resulted from nasal obstruction due to neglected sinus disease. The failures occurred in the first half of the series.

It is possible to tell by the fifth day whether the operation is successful. All patients in whom communication has been established

by that time have maintained it up to the present, a period in some instances of over three years.

From these observations it appears that in selected cases of chronic dacryocystitis, dacryocystorhinostomy is indicated because in a worth while number it will relieve epiphora and infection. If a few simple rules are observed, the operation is easy to perform, taking on an average of less than one-half hour. It can be done by the average ocular surgeon with the instruments found in the ordinary ophthalmic operating room.

570 Park Avenue.

ANGIOMATOSIS RETINAE

REPORT OF A CASE WITH A PATHOLOGIC STUDY OF THE ENUCLEATED EYE

ROBB McDONALD, M.D.*

AND

STUART W. LIPPINCOTT, M.D.

Assistant Instructor, Department of Pathology, University of Pennsylvania

PHILADELPHIA

Angiomatosis retinae was described as early as 1882 by Fuchs [1] and subsequently by several other investigators, but it was von Hippel [2] in 1904 who first recognized it as a clinical entity. Since then his name has been associated with the disease. Scarlett [3] reported the first case in this country in 1925. Cushing and Bailey [4] and Bedell [5] have subsequently reviewed the literature, but studies on an enucleated eye employing differential staining technics have not been recorded in the American literature and rarely in the literature of other countries. Because of this fact and the rarity of the condition, it has been considered worth while to report a case.

REPORT OF A CASE

A boy aged 15 years was first seen and admitted to the Wills Hospital, to the service of Dr. Frank C. Parker, on May 24, 1937. His parents stated that when he was 5 years of age he had been hit above the right eye with a stone. Shortly after this he lost all useful vision in that eye. When 11 years old he became aware of failing vision in his left eye. A local oculist prescribed glasses, and the patient carried on for four years. His vision became rapidly worse in August 1936, and he was admitted to the Mercy Hospital in Pittsburgh the same month. It was learned, while preparing this report, that the condition was diagnosed as angiomatosis retinae at that time by Dr. Rhodes and that the case was presented before the Pittsburgh Ophthalmological Society.[6] After the child's discharge from the Mercy Hospital, his vision became progressively worse, decreas-

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* Fellow in Ophthalmology, University of Pennsylvania.

1. Fuchs, E.: Aneurysma arterio-venosum retinae, Arch. f. Augenh. **11**:440, 1882.

2. von Hippel, E.: Ueber eine sehr seltene Erkrankung der Netzhaut, Arch. f. Ophth. **59**:83, 1904.

3. Scarlett, H.: Angiomatosis of the Retina, Arch. Ophth. **54**:183 (March) 1925.

4. Cushing, H., and Bailey, P.: Hemangiomas of the Cerebellum and Retina (Lindau's Disease), with Report of a Case, Arch. Ophth. **57**:447 (Sept.) 1928.

5. Bedell, A. J.: Angiomatosis Retinae, Am. J. Ophth. **14**:389 1931.

6. Rhodes, D. H.: Angiomatosis Retinae, Am. J. Ophth. **20**:522, 1937.

ing from 20/80 to perception of light on his admission to the Wills Hospital. The remainder of his personal and family histories, especially with regard to visual disturbances, was found to be unimportant.

Physical Examination.—The results of the physical examination were essentially negative, but the following findings are recorded because of their bearing on the case. The skin and mucous membranes were normal, with no evidence of nevi, papillomas or nevus flammeus. The neurologic examination did not reveal any evidence of organic disease. The spleen and kidneys were not palpable. The laboratory tests showed 4,590,000 red blood cells and 7,300 white blood cells (46 per cent neutrophils, 48 per cent basophils, 5 per cent eosinophils and 1 per cent monocytes); a coagulation time of three minutes; a negative Wassermann reaction of the blood; a negative reaction to the Mantoux test with purified protein derivative (0.000001 and 0.001 mg.) and normal urine. A roentgenogram of the right eye did not reveal any opaque foreign body.

Ocular Examination.—Right Eye: At the junction of the outer and the inner third of the eyebrow there was a small scar, the site of the wound from the stone. The lids, cilia, lacrimal passages, conjunctiva and sclera were normal. The eye was divergent about 20 degrees. The cornea was clear; the corneal reflex was not impaired. There was no evidence of any old penetrating wound. The anterior chamber was of normal depth; the iris was atrophic and was bound down to the anterior capsule of the lens, which was opaque. Examination with the slit lamp showed a few fine pigment deposits on the posterior surface of the cornea. The aqueous was clear. The iris was markedly thinned, and many new blood vessels were visible in the stroma; some of them could be seen to anastomose on the thickened anterior capsule of the lens. Transillumination of the eye was equal in all meridians and showed a red reflex in the pupillary space. The tension was 18 mm. (Schiötz). Vision was nil.

Left Eye: The lids, cilia and lacrimal passages were normal, and the external ocular movements were full. The conjunctiva and sclera were normal. The cornea was clear, with a normal sensory reflex. The anterior chamber was of normal depth. The pupil was round and reacted to light. The lens was clear. The media showed a faint haze, but there were no large opacities. The fundus presented a striking and bizarre picture. Most impressive were the changes in the vascular system. The disk could not be seen, but just above the posterior pole an enormously dilated vein was visible in the upper temporal quadrant. It coursed in a serpentine fashion over the many folds of the detached retina, the displacement of which forward increased temporally and below. The vein ended abruptly in a large reddish cyst in the extreme periphery about 1 disk diameter above the horizontal meridian. The vein was followed above by a similar dilated vessel of slightly smaller size; it was hidden in part of its course by the many folds of the retina, but it could be seen to enter the cyst just above the vein. There were two other cystlike dilatations, one above about 1 o'clock and another below at 7 o'clock. The artery in the latter could not be seen among the retinal folds.

The vessels, especially those communicating with the cysts, were enormously dilated and tortuous and had a peculiar reddish reflex which gave the impression that they were about to burst. The entire temporal and lower half of the retina was detached, the detachment being uneven, with many undulating folds. Its color varied from red to yellow, and in places it was of a greenish hue. No normal area was seen in the fundus. A small sector of retina above appeared to be attached with normal vessels, but the retina was yellow and atrophic. No areas of choroid or any choroidal vessels were visible. Examination with the slit

lamp showed the cornea to be clear, the aqueous to contain a few fine pigment granules and the iris to be normal. The lens was clear, and the vitreous contained many fine floating opacities.

The tension was 20 mm. (Schiötz). Vision was limited to perception of light, with projection limited to the nasal field.

The patient was hospitalized for five weeks while these procedures were carried out. He was given 1,405 mg. hours of radium over the left orbit, four treatments one week apart being given. There was no apparent change after the radium therapy. This therapy was instituted because it had been recommended by Bedell [7] and others but with the belief that this was an inherently benign tumor and the treatment would therefore be of no avail.

The patient was shown to several examining members of the American Board of Ophthalmology, and all but two concurred with our diagnosis of angiomatosis

Fig. 1.—Photograph of a painting of the fundus of the left eye showing the cyst-like dilatations in the periphery, the dilated vessels and detachment of the retina.

retinae. The case aroused considerable discussion, but to exclude the possibility of a malignant tumor of the blind right eye, the latter was enucleated on June 14. The patient was discharged from the hospital on July 3.

Macroscopic Report on the Right Eye.—The globe was of normal size. When it was sectioned, the anterior chamber was found to be deep and almost completely filled with a serofibrinous exudate. There were an occlusion and seclusion of the pupil, with an accompanying iris bombé. The lens showed cataractous changes, with many calcareous deposits, and was surrounded posteriorly by an

7. Bedell, A. J., in Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 779.

organized exudate, which had caused complete detachment of the retina. The subretinal space occupying almost two thirds of the posterior chamber was filled with a serous exudate.

Microscopic Report on the Right Eye.—The cornea showed practically no change except at the limbus, where there were many new blood vessels and a moderate degree of round cell infiltration. There was some degeneration of the endothelium. The anterior chamber was deep, and at the filtration angle there was a complete anterior synechia. The canal of Schlemm was completely obliterated. The pupillary space was filled with an organized exudate; there was seclusion of the iris by posterior synechia, and the iris was pushed forward into the anterior chamber, producing iris bombé.

The iris and ciliary body showed loss of cellular structure, with fibrous tissue proliferation and many newly formed blood vessels. The pigment layer, though atrophic, was intact. The posterior chamber was deep and filled with a serofibrinous exudate.

The lens was surrounded by a fibrinous exudate and showed advanced hyaline and calcareous changes. The entire retina was detached and extended from the posterior surface of the lens to the optic nerve as a V-shaped band. Anteriorly it merged with the atrophic ciliary processes by many strands of connective tissue. The normal retinal structure had been replaced by the tumor. The tumor lay in this stalk of tissue extending from the posterior pole to the lens, and a large dilated vessel could be seen extending almost its entire length. Many large cyst-like dilatations could be seen lined by endothelium; some of them contained red blood cells. The whole structure was permeated by thin strands of connective tissue and many newly formed blood vessels.

The choroid had been almost completely replaced by fibrous tissue, but a few dilated blood vessels were visible posteriorly. Anteriorly the choriocapillaris was barely distinguishable, and there was some round cell infiltration. On both sides of the nerve head there were areas of bone formation lying in the band of fibrous tissue that had replaced the choroid.

The sclera appeared normal. There was some dipping of the lamina cribrosa, but the nerve head itself did not show any change. The intervaginal space was moderately well filled.

Microscopic Report on the Tumor of the Right Eye.—The section of tumor stained with hematoxylin and eosin did not present a uniform appearance. The matrix was cellular, and under low power magnification this background, which was chiefly glial, was most prominent. It appeared to form the major part of the tumor. The Bielschowsky stain accentuated the glial framework, the cells and fibers of which stained deep gray while the reticulum appeared as a fine black webbing sparsely distributed. Similarly, with the Holzer stain glial tissue was recognized by the deep blue-staining fibers. These fibers occasionally formed a dense network, although individual, smaller wavy fibrils could be seen arising from one or both poles of elongated and deeply nucleated cells. While the glial tissue formed the greater part of the background, it assumed no definite arrangement. It was compact or loosely arranged in whorls. These whorls, however, should not be mistaken for rosette formations, for the latter do not occur, nor does actual palisading of cells as is seen in tumors of neurogenic origin.

In addition to the typical and odd monster glial cells and fibers, there were other far less numerous and conspicuous cellular elements. These could be seen to advantage by the toluidine blue method of staining, which sharply delineated them. Slender, flat endothelial cells could be seen, and in one focal area they

appeared in a bizarre pattern of crosses and chains of closely packed cells. In the less dense cellular areas, ovoid, vesicular nucleated cells were found. Polygonal cells with central nuclei were seen. Occurring but rarely were ganglion cells, some of which were degenerating, and mast cells with red granules in their cytoplasm. Isolated peripherally was a group of cholesterol crystals with foreign body giant cells about them, but no pseudoxanthoma cells were noted. Blood pigment and retinal pigment were observed in small deposits.

Even though these cellular components seemed to constitute the greater part of the tumor, other structures attracted attention. Of these, most striking were the

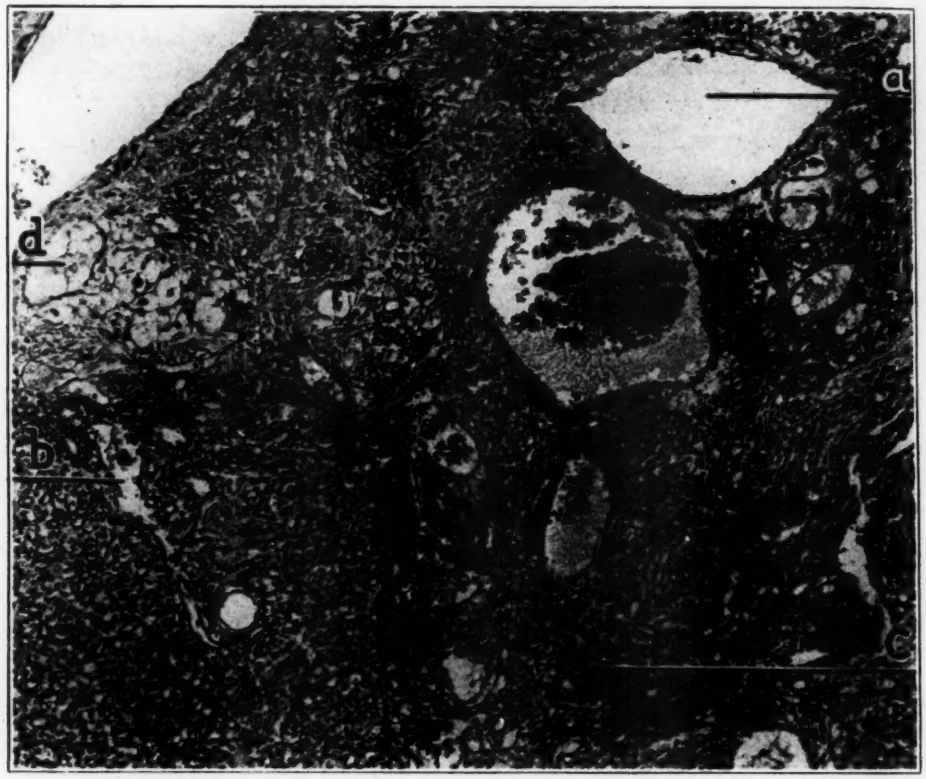

Fig. 2.—Low power magnification of a section of the tumor of the right eye, showing cysts (*a*); capillaries (*b*); connective tissue (*c*), mostly glial; and degenerative connective tissue (*d*). Hematoxylin and eosin; × 112.

cystic dilatations, the largest of which occupied a low power field. Many were much smaller and more perfectly circular. They were completely denuded of lining cells, which might be due either to pressure over a long period by the contained pink-staining material or to simple degeneration. Of great importance were the capillaries that characteristically were long and narrow and frequently had red blood cells within them. They occasionally appeared like sinusoids when cut transversely. Their walls were formed by tapering slender cells. Not uncommonly the entire lumen was filled by roughly circular layers of endothelial cells,

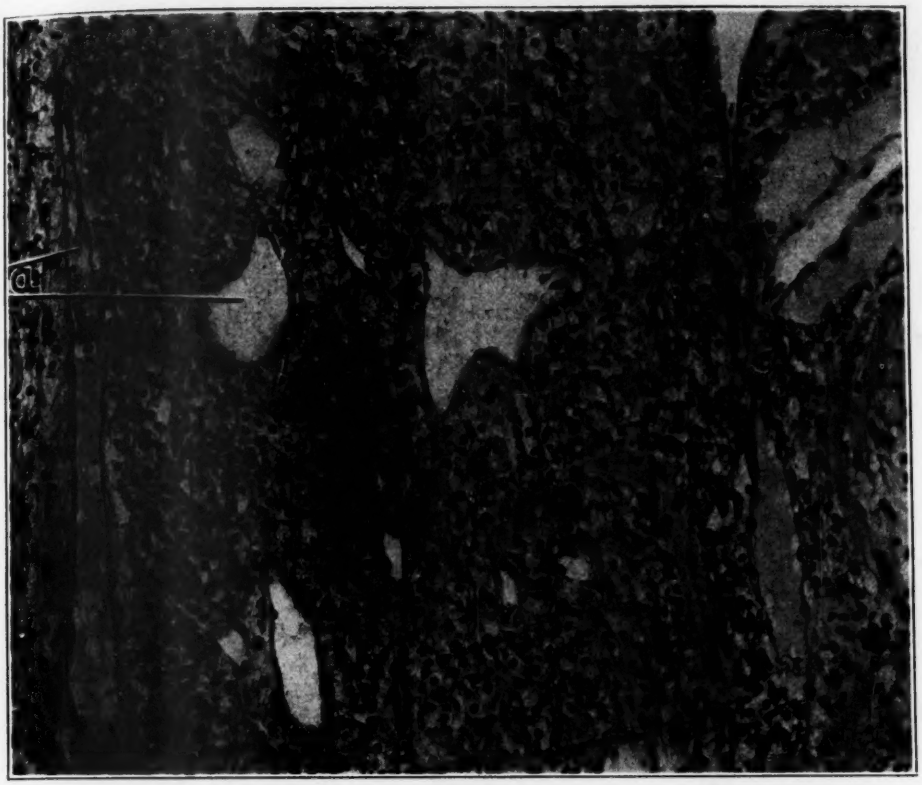

Fig. 3.—Section showing the variation in size and shape of the capillaries (*a*) in the tumor. Hematoxylin and eosin; × 220.

Fig. 4.—Section showing the cellular background in which can be distinguished endothelial lining cells of a capillary (*a*), glial cells with tapering fibers (*b*), fibroblast (*c*), a degenerating ganglion cell (*d*) and red blood cells (*e*). Toluidine blue; × 436.

which as they approached the center became more plump but did not exhibit any mitotic figures. About the cystic dilatations and larger capillaries, the Masson stain showed circular bands of green fibers. They did not penetrate into the substance of the tumor but coiled about the vessels and were composed of collagen and connective tissue. In studying the section stained by the Van Gieson method for further evidences of collagenous connective tissue fibers, the same distribution as was seen in the preparation stained by the Masson method was here recognized by the deep red fibers. Furthermore, smaller red fibers wove delicately between some of the more dense glial fibers.

Fig. 5.—Section showing the deeper staining collagenous fibers about the vessels and woven in the matrix. Van Gieson's stain; × 112.

In reviewing the structural composition of the tumor, it was evident that the background was primarily composed of glial cells and fibers with but little intertwining collagenous connective tissue. Enmeshed in this matrix were cystic dilatations and numerous capillaries. Other varied, but infrequent, cellular elements were present. It is our belief, however, that the initial event was the formation of the angioblastic derivatives, represented by the cystic dilatations and capillaries and that the gliosis was secondary and of long standing. This, then, was not a tumor of glial derivation but a tumor of vascular origin which should be considered as a congenital malformation.

COMMENT

We have presented a case of angiomatosis retinae. The diagnosis was made in the left eye on the ophthalmoscopic examination and confirmed by histologic examination of the blind right eye. The early onset of the disease is interesting, having been present in the right eye apparently since the child was 5 years of age. It is doubtful whether the injury had any bearing on the case, except to attract attention to the loss of vision in the right eye.

The clinical diagnosis in this case was either angiomatosis retinae or Coats's exudative retinitis. The clinical differentiation is not always easy. Both diseases are characterized by extensive subretinal exudate and subsequent detachment of the retina, but the singular changes in the retinal vascular system in this case favored a clinical diagnosis of angiomatosis retinae.

Characteristically in the disease, dilated vessels first appear, running to a cystlike formation or aneurysm in the periphery of the fundus. Later, secondary changes occur, namely, exudation, hemorrhages, detachment of the retina, iridocyclitis, secondary glaucoma, cataract and finally hypotension with phthisic changes.

The enucleated eye shows the secondary features. The tumor of the right eye has been shown to be of vascular origin, representing a congenital malformation, even though the gliosis is so marked that it nearly masks the true nature of the derivation of the tumor.

LESION OF THE OPTIC TRACT

PROBABLY THE RESULT OF INFECTED SPHENOID SINUSES

ALBERT N. LEMOINE, M.D.
KANSAS CITY, MO.

The article of Onodi entitled "The Optic Nerve and the Accessory Cavities of the Nose" [1] in 1908 laid the anatomic foundation for the theory of blindness as a result of disease of the accessory sinuses. Smith, Van de Hoeve and Loeb reported the clinical application of this theory and some results obtained by surgical intervention on the ethmoid and sphenoid sinuses. White [2] published reports of 2 cases of retrobulbar neuritis due to disease of the accessory sinuses in 1916 and of 3 cases in 1917. These articles stimulated a wave of enthusiasm, with the result that most cases of retrobulbar neuritis not due to purely exogenous toxins, such as tobacco, alcohol, lead, thallium acetate, etc., were attributed to disease of the posterior sinuses. Since that time in this country the pendulum has swung back and forth from disease of the posterior sinuses to multiple sclerosis or foci of infection as being the predominating etiologic factor in these cases. European ophthalmologists constantly attributed most of these cases of retrobulbar neuritis to multiple sclerosis.

A case in which the lesion was similar to retrobulbar neuritis but was back of the chiasm is reported here. The findings and course of the disease suggest that sphenoid sinusitis was the cause of the condition.

REPORT OF A CASE

B. B. S., a married man aged 39, who had no children, first consulted Dr. V. H. Bergmann on Dec. 18, 1937, complaining of a dull pain about the waist extending to the knees. He had an enlargement of the prostate, with a slight amount of residual urine that had made it necessary for him to get up on an average of twice a night since 1932. All this time he had been under the care of a urologist. In 1935, at the age of 37, he became impotent. The past history preceding this trouble was essentially unimportant.

Read at the Seventy-Fourth Annual Meeting of the American Ophthalmological Society, San Francisco, June 10, 1938.

1. Onodi: The Optic Nerve and the Accessory Cavities of the Nose, Ann. Otol., Rhin. & Laryng. **17**:1, 1908.

2. White, L.: Loss of Sight from Retrobulbar Neuritis Due to Accessory Sinus Disease, with Report of Two Cases, Boston M. & S. J. **174**:790, 1916; Loss of Sight from Posterior Sinus Disease, with Report of Three Cases, ibid. **176**:891, 1917.

Laboratory and physical examination at that time gave entirely negative results except that transillumination of the sinuses showed a cloudy right antrum, and rectal examination revealed "enlargement of the prostate with the right lobe firmly bound down and slight fibrosis. The left lobe and central raphe were full and indurated." The diagnosis was chronic prostatic hypertrophy. Dr. Bergmann [3] expressed the belief that most of these cases are due to a deficiency of the anterior lobe of the pituitary gland, so the patient was given 1 cc. of anterior pituitary extract every four days.

By December 31 the patient noticed definite improvement in his well-being, emotional state and sexual outlet. This improvement continued until Jan. 14, 1938, the time of the onset of the present illness.

On January 14 pain developed in the right eye which was aggravated when the patient moved his eyes. Five days later a blurring of the vision developed, most marked in the right eye. The patient consulted an oculist, who concluded that the condition was due to multiple sclerosis and gave him 15 grains (0.97 Gm.) of potassium iodide three times a day. At this time a careful neurologic examination gave entirely negative results. Complete laboratory examination revealed the following values: blood sugar, 89.2 mg. per hundred cubic centimeters; nonprotein nitrogen, 28.2 mg.; creatinine, 2.1 mg.; chlorides, 4.17 mg.; cholesterol, 152.2 mg.; uric acid, 3.6 mg.; red blood cells, 4,590,000; white blood cells, 8,000; hemoglobin, 90 per cent; polymorphonuclear cells, 72 per cent; nonfilamented forms, 6 per cent, and large and small lymphocytes, 22 per cent.

The impairment of vision gradually became worse. The patient was referred to me on January 29. At this time vision in the right eye was limited to counting fingers at 1 foot (30 cm.); vision in the left eye was 20/60 + 2. The right pupil was larger than the left and reacted sluggishly to light. The left pupil reacted sluggishly to light. The fundi were normal with the exception of a slight temporal pallor of the disk and slight blurring of the margins of the disk. Both fields showed a homonymous defect of the left inferior quadrant (fig. 1) involving the maculas. Roentgenographic studies were ordered. Ephedrine sulfate was instilled into the nose; the instillation was repeated two days later. On the fourth day the sector defect (fig. 2) was much smaller, and the vision improved to 6/400 in the right eye and 20/20 — 1 in the left eye.

The roentgenographic examination, made by Dr. E. W. DeWeese (fig. 3), revealed the following facts:

The examination included the entire head and revealed no demonstrable intrinsic disease of the bone or secondary manifestations of an intracranial lesion. The inner and outer tables of the skull were smooth and regular, and the calcium content was not locally or generally impaired.

There was no exaggeration of the vascular markings over the inner table or abnormal widening of the suture lines.

The sella turcica was unusually small, and the clinoid processes were much flattened. Another unusual finding was the degree of development of the sphenoid sinuses. There was an aerated cellular structure extending around the sellar fossa on all surfaces with the exception of the dorsal. This was obviously a developmental variation. No true erosion of the osseous structure of the sphenoid walls was found, nor were there definite pathologic changes within the cells or the clinoid processes.

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3. Bergmann, V. H.: Prostatic Hypertrophy as a Definite Endocrine Problem, Missouri State M. J. **34**:119, 1937.

Further roentgenographic studies to include the paranasal sinuses showed large, well formed cells, symmetric in outline. No gross blocking opacities of any of the cells were found. There was present a definite mucocele within the right antrum, and the sphenoid sinuses did not appear to be normally ventilated. There was also a rather marked general increase in the intranasal tissue, with the turbinates apparently much hypertrophied. There were some thickening and mild deviation of the nasal septum to the right. A questionable small spur formation was present on the nasal septum on the right side.

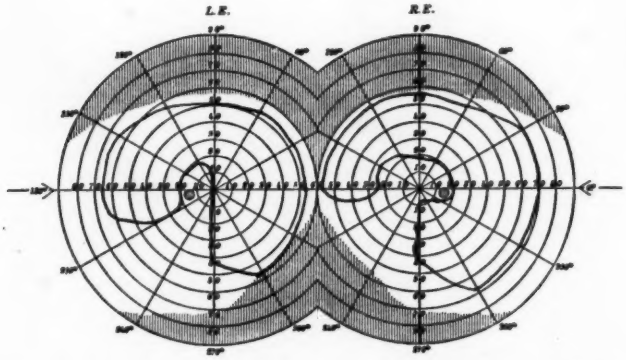


Fig. 1.—Peripheral fields of vision on Jan. 29, 1938, the day of the first examination. Vision in the left eye was 20/60 + 2; that in the right eye was limited to counting fingers at 1 foot (30 cm.). A 1 degree target was used.

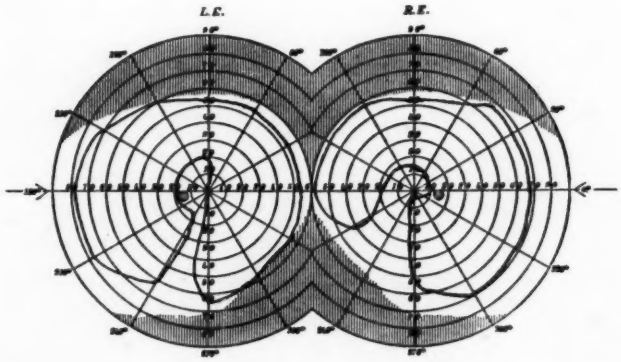


Fig. 2.—Peripheral fields of vision on Jan. 31, 1938. There was improvement due to shrinking the mucous membrane of the nose with ephedrine sulfate. Vision in the left eye was 20/20 — 1; that in the right eye was 6/400. A 1 degree target was used.

The roentgenographic picture was interpreted as being indicative of a significant pathologic process within the sphenoid cell.

After February 2 the vision rapidly failed, so that on February 4 the vision of the left eye had become so poor that it was difficult for the patient to get around. He was examined by Dr. Paul Lux (rhinologist), who reported that there was no pus or crusting in the nasal passages and no change in the nasal mucosa.

The vision rapidly became more impaired, and on the evening of February 7, when the patient went to the hospital, he could not see his bed.

The hematologic picture had been undergoing a gradual change in the number and proportion of the white cells from the time the patient was first examined, as indicated by the following findings:

On January 19 the hemoglobin content was 90 per cent. There were 4,590,000 red blood cells and 8,000 white blood cells, with 73 per cent polymorphonuclear cells and 27 per cent large and small lymphocytes.

On January 31 there were 9,700 white blood cells, with 78 per cent polymorphonuclear cells and 22 per cent large and small lymphocytes.

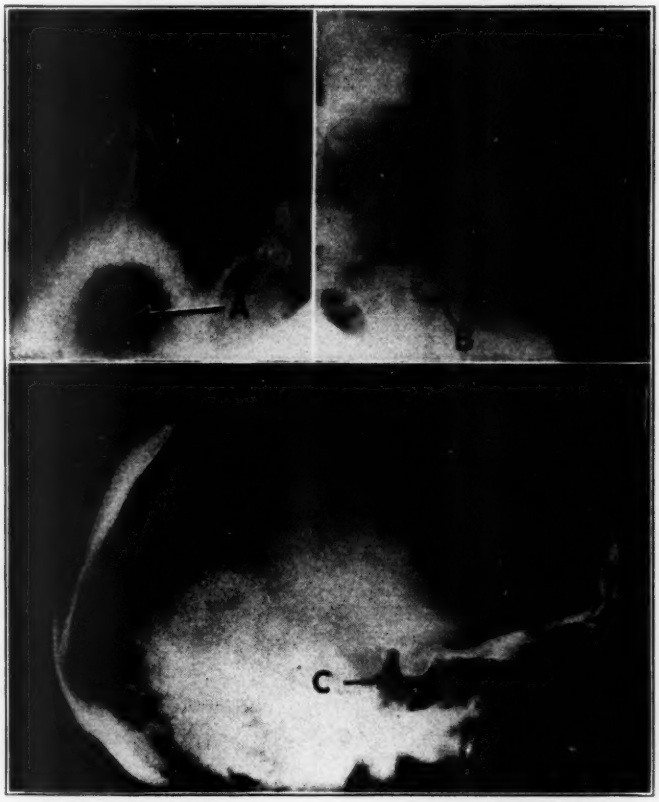


Fig. 3.—*A*, anterior posterior view of the sphenoid sinuses taken through the mouth. Note the definite pathologic process as revealed in the veiled opacities. *B* shows the marked blocking of the nasal passages caused by general hypertrophy of the turbinates. *C* shows the aerated sphenoid sinus extending into the posterior clinoid process with a veiled opacity.

On February 7 there were 10,600 white blood cells, with 81 per cent polymorphonuclear cells and 19 per cent large and small lymphocytes.

It will be noted that the white blood cells and the polymorphonuclear cells were gradually increasing.

In view of the roentgenographic picture, coupled with the changes in the fields that indicated that the lesion was in close proximity to the nasal pathologic

process, and the rapidly failing vision of twenty-one days' duration, associated with hematologic changes that pointed to increased activity of infection, I recommended draining the sphenoid sinuses.

On February 8 Dr. Lux performed a submucous resection and a radical operation on both sphenoid and ethmoid sinuses; a window was made in each of the antral walls under the corresponding inferior turbinate.

Operation revealed a thickening of the mucoperiosteal lining of both sphenoid sinuses with a hemorrhage into the mucoperiosteal lining of the posterior superior wall of the right sphenoid sinus. There was no pus in either sinus. The bony walls

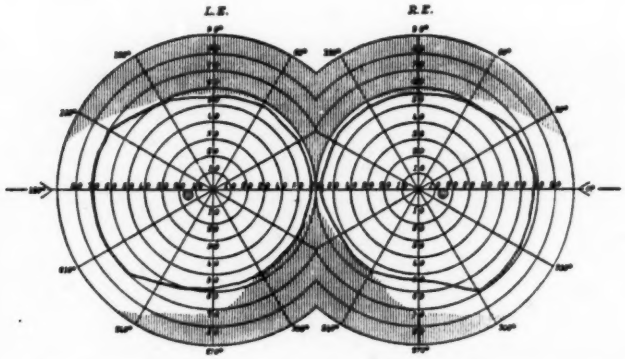


Fig. 4.—Peripheral fields of vision thirteen days after operation (Feb. 21, 1938) on the sphenoid sinuses. Vision in the left eye was 20/20 — 2; that in the right eye was 20/200. A 1 degree target was used. There was concentric contraction to all colors.

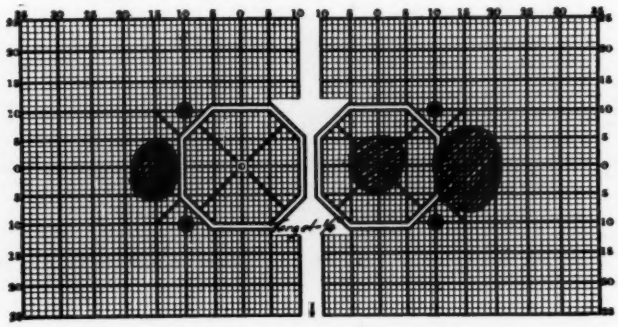


Fig. 5.—Peripheral fields of vision thirteen days after operation.

of both anterior and posteroir ethmoid cells on the right and left sides were destroyed, probably the result of a chronic catarrhal process. There was no visible change in the mucoperiosteal lining of the antrums.

The day after the operation the vision was much better, and on the second postoperative day (February 10) the patient could read large print. His vision rapidly improved.

On February 21 vision in the right eye was 20/200 and in the left eye 20/20 — 2. The sector defect (fig. 4) in the fields had disappeared, but a large central scotoma was still present in the right eye (fig. 5).

On March 12 the vision in the right eye was 20/30 — 2; that in the left eye was 20/20 — 2.

The fundi were normal except a slight pallor of the temporal quadrant of the disks. The peripheral fields had returned to nearly normal with a small pericentral scotoma.

On February 12 the hemoglobin content was 96 per cent; there were 4,710,000 red blood cells and 7,900 white blood cells, with 65 per cent polymorphonuclear cells, 32 per cent lymphocytes and 3 per cent eosinophils.

The patient was given 1 cc. of anterior pituitary extract every five days after the operation. On March 28 vision in the right eye was 20/30 +; that in the left eye was 20/15 — 2. The visual fields were normal. The blindspot in the right eye enlarged 1 degree (fig. 6), and it required a 1 degree target to recognize green centrally with the right eye. The patient was then sexually normal and had no nocturia. There was a marked diminution in the size of the prostate.

On May 25 vision in the right eye was 20/20; that in the left eye was 20/15 — 1. The peripheral fields and the blindspots were normal, and the patient could recognize colors in each eye with a ½ degree target. He had received no treatment since March 28 and stated that he felt better in every respect than he has since 1932.

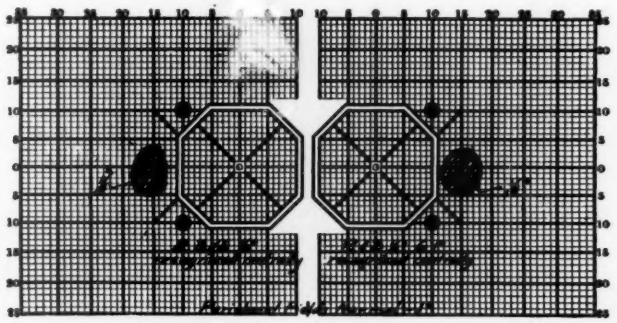


Fig. 6.—Peripheral fields of vision twenty days after operation on sphenoid sinuses. The only defect was 1 degree of enlargement of the right blindspot. Green was recognized only with a 1 degree target.

COMMENT

In this case hypertrophy of the prostate developed in a healthy person at the age of 33 and impotency at the age of 37. This condition was attributed by an endocrinologist to a deficiency of the anterior lobe of the pituitary gland. Improvement was taking place with the administration of anterior pituitary extract, when a lesion of the right optic tract developed, which progressed to complete loss of central vision in twenty-one days. The failing vision was temporarily improved by the use of ephedrine in the nose, as had been observed by Frost.[4] The roentgenograms revealed poorly aerated sphenoid sinuses extend-

4. Frost, A.: Papilledema, with Special References to Papilledema Associated with Sinus Disease, Tr. Am. Ophth. Soc. **33**:480, 1935.

ing into the posterior clinoid processes. At operation the right sphenoid sinus was found to have a hemorrhagic area on the posterior wall, which was the portion extending into the posterior clinoid process and in close proximity or possibly in apposition to the superior portion of the right optic tract involving the fibers corresponding to the defect in the visual field, according to the scheme of Traquair.[5]

After the operation the vision and fields of vision returned to normal, and with six weeks of therapy with anterior pituitary extract the patient returned to sexual normality with no recurrences to date.

The logical explanation of this picture is that the patient had chronic sinusitis in an anomalous sphenoid sinus that extended into the posterior clinoid process back of the optic chiasm, causing a retrobulbar neuritis of the right optic tract. The modus operandi of involvement of the optic tract is problematic. It might have been the result of an actual invasion of the tract by a virus or toxins by direct continuity.

Since there was improvement temporally in the defects of the visual fields and the vision after shrinking the mucous membrane of the nose and since the disease progressed rapidly with an equally rapid improvement immediately after the opening of the sphenoid sinuses, it is not likely that the condition was due to thrombi, as was pointed out by Putnam.[6]

If the whole process had been due to invasion of the optic tract by a virus and if the theory is accepted that multiple sclerosis is due to a virus, it is possible that the infection will again become active and that multiple sclerosis will develop in the future. However, if one accepts the theory of thrombi or toxins coming from the sphenoid sinus, the patient will most likely remain permanently cured.

I feel that all patients with retrobulbar neuritis should be thoroughly studied, especially those in whom the condition seems to be secondary to disease of the posterior nasal sinuses. Such studies may lead to the solution of the perplexing problems of the underlying causes of multiple sclerosis and other demyelinating diseases.

DISCUSSION

DR. A. J. BEDELL, Albany, N. Y.: I wish to call attention to the observation of choked disk in 3 cases of arachnoiditis. (Slides were used to demonstrate the appearance of the fundi in the 3 cases.) Examination of a 12 year old girl revealed pale disks with indistinct margins, surrounded by retinal edema, retinal hemorrhages and exudates. Before operation the vision was reduced to 2/200. Recovery

5. Traquair, H. M.: An Introduction to Clinical Perimetry, ed. 2, London, Henry Kimpton, 1931, p. 66.

6. Putnam, T.: Etiological Factors in Multiple Sclerosis, Ann. Int. Med. **9**: 854, 1936.

was complete without defects in the visual field. Ultimate vision was better than 20/15.

A man, aged 39, complained of headache. Examination showed swollen disks and retinal hemorrhages and exudates. Recovery after operation was complete, with full restoration of the visual fields and normal visual acuity.

In a 17 year old boy examination showed swollen disks and retinal exudates. After operation, which revealed arachnoiditis, vision was fully restored.

Dr. Albert D. Frost, Columbus, O.: I observed 2 cases of arachnoiditis two years ago and made my studies the subject of my thesis presented to the American Ophthalmological Society. In these cases papilledema and loss of vision were accompanied by headache. The symptoms were relieved by shrinkage of the nasal mucous membrane. Full restoration of vision was accomplished by repeated shrinkage of the nasal membrane and the opening of the sphenoid and ethmoid sinuses. The first response to the nasal treatment was improvement of vision and reduction of the size of the blindspot within thirty minutes. The swelling of the disk disappeared within three days.

Dr. Derrick Vail, Cincinnati, O.: I agree fully with Lemoine. The remarkable case that he reports supports with overwhelming logic and evidence the premise that the sphenoid sinuses and chiasmal cisterns have a close relationship, much closer than has been realized in the past. Whether this relationship is entirely vascular or lymphatic remains to be demonstrated. It likewise remains for the future to determine why opening and draining the sphenoid sinuses in cases of retrobulbar optic neuritis have resulted in so many instances of dramatic restoration of vision, as reported in the literature. In 1930 Cushing, discussing the chiasmal syndrome (Arch. Ophth. 3:704 [June] 1930), stated "that a mild meningeal reaction may be produced in the cerebellopontile cisternae by an inflammatory process in the middle ear with symptoms suggesting those of an acoustic tumor, is well known. There is no reason, therefore, why a similar process might not occur in the chiasmal cistern in association with inflammatory processes in the accessory paranasal sinuses." The clinical significance, therefore, of Dr. Lemoine's case assumes a great importance. Here is a patient who exhibits definite pituitary dystrophy, characteristic defects in the visual field and an extraordinary condition of the sphenoid sinuses, as shown roentgenographically. Exploration showed an absence of pathologic material in the sphenoid sinuses, except for a hemorrhagic area on the posterior wall of the right sphenoid sinus, which was the portion extending into the posterior clinoid process. The temporal pallor, the slight blurring of the margin of the disk, the characteristic sector defects and the rapid improvement of symptoms following nasal drainage are to be carefully considered as evidence pointing toward the chiasmal cistern as the seat of the disturbance. This significance will not be lost if multiple sclerosis were to develop later.

EVENTS OF VASCULARIZATION AND DEVASCULARIZATION SEEN IN CORNEAS

P. F. SWINDLE, PH.D.
MILWAUKEE

The Rocky Mountain bighorn sheep (Ovis canadensis Shaw) is the only animal, so far as my observations go, in which the apparently normal cornea becomes extensively invaded by blood vessels. This animal, like others, has the pericorneal network, but at or about the time of birth a number of bouquets of vascular loops (figs. 14 and 15) develop between various corneal lamellae. Each of these bouquets originates from a scleral arteriovenous anastomosis which loops into the cornea (figs. 8 and 9) and then metamorphoses into a principal artery, an accompanying vein, many paired arterial and venous branches and many capillaries which connect the tips of the smallest arteries with the tips of the smallest veins (figs. 14 and 15). The corneal tissue remains extremely clear during the invasion of it by the scleral arteriovenous anastomoses. It is possible, therefore, to observe the metamorphosis of these anastomoses in the cornea of the lamb during early postnatal life.

The metamorphosis of any of the arteriovenous anastomoses in the cornea of the lamb does not cease until the lumen of the principal arterial trunk becomes spontaneously obliterated. This obliteration can be observed from time to time at various parts of the cornea during the very early postnatal life of the animal. The vascularization of the cornea takes place rapidly, and the bouquets of vascular loops vanish rapidly after the lumens of the principal arterial trunks become obliterated. The vascularization ceases four or five days after birth. The corneal tissue is extremely clear during the entire time of the vascularization and devascularization of it.

Other corneas may become vascularized if they become inflamed (figs. 1 to 7 inclusive). In cases in which there are some persisting scleral arteriovenous anastomoses these vessels may loop into the inflamed corneal tissue, and the metamorphosis of these anastomoses may then be either identical or almost identical with the metamorphosis of the arteriovenous anastomoses in the apparently normal cornea of the lamb. For instance, such bouquets of vascular loops as those in the inflamed cornea of the black panther (fig. 5) are similar to the most

From the Department of Physiology, Marquette University School of Medicine.

extensively developed ones in the cornea of the lamb (figs. 14, 15, 16 and 17). In the panther's cornea, however, the capillaries of the various bouquets of vascular loops united to form a general network of capillaries. A general capillary network uniting two or more bouquets with one another was not found in the corneas of any of the lambs. I shall accordingly speak of the complete metamorphosis of the scleral arteriovenous anastomoses, as in the panther's cornea, and of the incomplete metamorphosis of the scleral arteriovenous anastomoses, as in the lamb's cornea. The metamorphosis of these anastomoses in a tissue is not complete until or unless capillarization progresses to such a stage that a general network of capillaries exists.

In the absence of scleral arteriovenous anastomoses corneas may also become vascularized if they become inflamed (figs. 1, 2, 3, 4 and 7), but the bouquets of vascular loops, like those in figures 5, 14 and 15, cannot be found in these corneas. For instance, the inflamed cornea of the adult sheep may become extensively vascularized, but bouquets of vascular loops, like those in figures 5, 14 and 15, cannot be found in it. The probable reason is that the supply of scleral arteriovenous anastomoses became exhausted during the original vascularization of the apparently normal cornea. In other animals, such as the cat, the supply of scleral arteriovenous anastomoses may become exhausted during a first vascularization of the cornea (fig. 5), so that no bouquets of vascular loops can be found in the tissue at a later vascularization of it. In the absence of scleral arteriovenous anastomoses an inflamed cornea may become invaded by preexisting blood vessels (figs. 1, 2, 3, 4 and 7), which are normally located at and near the limbus. This is a vicarious vascularization of the cornea, and it is a poor imitation of the much more systematic vascularization by the scleral arteriovenous anastomoses. Each scleral vessel, or in some instances each plexus of scleral vessels, which vicariously invades the cornea might appropriately be called a substitute for a scleral arteriovenous anastomosis, but it is a poor substitute because the cornea is often scarified and shrunken after the vessels in it vanish. A cornea may be entirely normal, however, after it has been extensively vascularized by scleral arteriovenous anastomoses and the corneal vessels have vanished.

In some earlier papers the metamorphosis of arteriovenous anastomoses was discussed principally in connection with the vascularization of the nasal lining and ear,[1] the nasal lining and brain [2] and the brain

1. Swindle, P. F.: The Architecture of the Blood Vascular Networks in the Erectile and Secretory Lining of the Nasal Passages, Ann. Otol., Rhin. & Laryng. **44**:913, 1936.

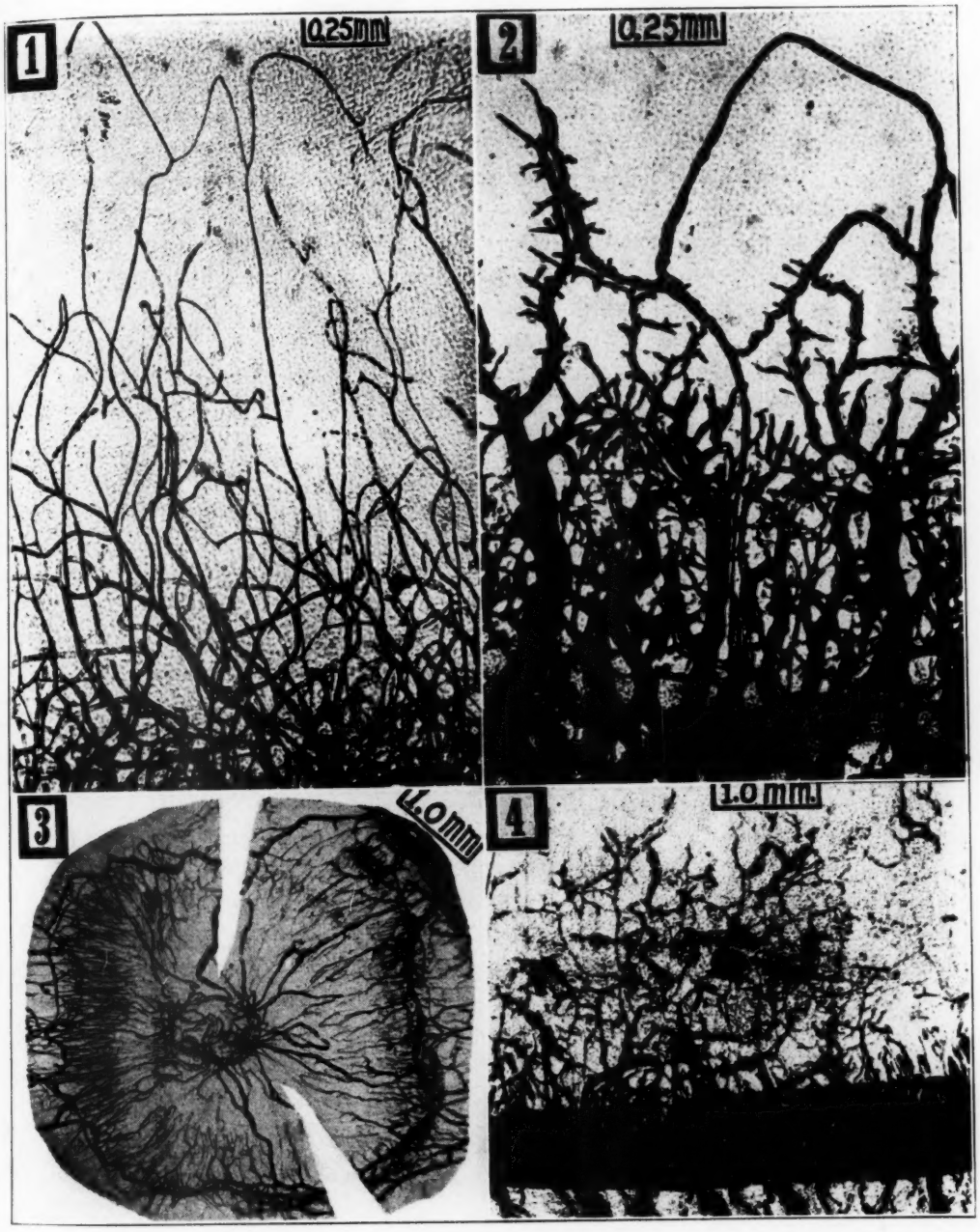
2. Swindle, P. F.: Nasal Blood Vessels Which Serve as Arteries in Some Mammals and as Veins in Some Others, Ann. Otol., Rhin. & Laryng. **46**:600, 1937.

Explanation of Plate

Figs. 1 to 4.—Figures 1 to 3, sections from the inflamed eyes of adult albino guinea pigs. The inflammation was caused by injecting a small amount of red mercuric sulfide (in water) into the vitreous humor. Each of the eyes was observed almost constantly for several hours each day after the first signs of inflammation were detected. Each of the animals was killed by first anesthetizing it with ether and then severing the abdominal aorta. India ink was then injected rhythmically into the peripheral stump of a common carotid artery until either all or the greater number of the scleral and corneal vessels were filled with the ink. These vessels were observed with a binocular microscope during the injection. The animal was then suspended, head down, for about four hours before the eye was removed.

In figure 3 three lines of invading loops of blood vessels can be seen. The crests of the loops of the first line met at the center of the cornea. No loops of the second line extend entirely to the center, and the loops of the third line (the large vessels of the circle of Hovius) extend only short distances into the cornea at only some places. Figure 1 shows principally the vessels of the first line of invasion. Figure 2 shows loops of the first line of invasion and also many of the scleral vessels which would have looped into the cornea and been recognized as the loops of the second line of invasion if the animal had lived. Figure 2 also shows many stumps of vessels which restrained the movement of the vessels of the first line of invasion for a certain time and then broke and permitted some loops to jump farther toward the center of the cornea.

Figure 4, section from the left eye of a half-grown wallaroo kangaroo (Macropus robustus Gould). On gross examination the animal seemed to have actinomycosis, but the fungus could not be found. Principally the throat and the left side of the head and neck were involved. The animal was killed by quick decapitation after it lay in a profound comatose state for five days in my laboratory. The right eye appeared to be normal. The conjunctiva of the left eye was severely inflamed, and a light gray substance was deposited irregularly on the inner surface of the cornea, but the corneal tissue was clear. Blood cells could be seen circulating in some narrow and broad channels deep in the cornea at various points near the limbus. The circulation was at times erratic and at times steady. The vessels were observed for several hours each day for four days. As soon as the animal was killed, india ink was injected rhythmically into the left common carotid artery while the other severed vessels in the neck were clamped with hemostats. Before the specimen was mounted, the uvea, some of the outer lamellae of the cornea and some of the superficial tissue of the sclera were removed, so that the deep vessels at and near the limbus could be seen more clearly. The portion selected for mounting was one in which some of the blood vessels had been carefully observed while the animal was living. The dark horizontal band in the lower part of the figure is a picture of the highly developed anterior portion of the cavernous plexus of scleral blood vessels (Swindle, P. F.: The Principal Drainage Channels of the Eye, Arch. Ophth. **17**:420 [March] 1937). The vessels in the cornea originated from this cavernous plexus. They lie between the membrane of Descemet and the posterior lamella of the cornea. The left cerebral hemisphere was found to be almost completely devascularized.



Figures 1-4.

and epiphyses.[3] The vascularization of the cornea by scleral arteriovenous anastomoses is similar to that of the brain and epiphyses. It is also similar to the final stage of the vascularization of the nasal lining and various other parts of the body. The vicarious vascularization of a cornea by substitutes for arteriovenous anastomoses is similar to the vicarious vascularization of necrotic areas at various parts of the body where these anastomoses do not exist.

At the 1933 session of the American Medical Association I exhibited some corneas which had become partially vascularized by substitutes for arteriovenous anastomoses. Some of the preexisting vessels, substitutes for these anastomoses, at and near the limbus were observed merely to move or loop toward the center of the cornea as they grew in length. Brückner [4] and Kreiker [5] had already called attention to this type of invasion of corneal tissue by blood vessels. I spoke of "vascularization without neovasculogenesis." Some other corneas shown at the same meeting illustrated a combination of "vascularization with and without neovasculogenesis." At the 1934, 1936 and 1937 sessions of the Wisconsin State Medical Society and at the 1938 session of the North Central Section of the American College of Surgeons I exhibited some of the specimens and also some others. In addition to the terminology used in 1933, I spoke of the "complete and incomplete metamorphosis of arteriovenous anastomoses and some of the substitutes for arteriovenous anastomoses into small arteries, small veins and capillaries."

The inflamed corneal tissue is rarely clear enough that the events of vascularization and devascularization can be clearly seen at all parts of the pannus. It is tempting, therefore, to examine such pathologic corneas only or primarily after they have been injected and cleared. By examining the vessels in the dead cornea only, or even by examining the living cornea for only a short period each day or each week, it is often impossible to differentiate between some of the generative and degenerative events. There is a disconcerting confusion of these events in the literature. In order to avoid confusing observations with assumptions, I shall describe only the events which were seen. The photomicrographs presented as figures will serve to show certain features of the corneal vessels and to facilitate the descriptions of the observed events of vascularization and devascularization of normal and inflamed corneas.

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3. Swindle, P. F.: Occlusion of Blood Vessels by Agglutinated Red Cells Mainly as Seen in Tadpoles and Very Young Kangaroos, Am. J. Physiol. **130:** 59, 1937.

4. Brückner, A.: Klinische Studien über Hornhautgefässe, Arch. f. Augenh. **62:**17, 1909.

5. Kreiker, A.: Die Entwicklung der Gefässbildung in der Hornhaut an der Hand von Spaltlampenbeobachtungen, Ztschr. f. Augenh. **50:**115, 1923.

MATERIALS AND GENERAL METHODS

The specimens herein presented as figures were selected from a large number which were collected during the past fourteen years. During this time I have performed autopsy on the animals which died in the Washington Park Zoo in Milwaukee and have had many opportunities during these years to study various normal and ailing animals under favorable conditions at the zoo. For instance, the events of both vascularization and devascularization of the cornea were observed in four healthy lambs, and the corneal vessels were studied in prepared specimens from three late fetuses and two early postnatal lambs. A number of animals from the zoo were treated and incidentally studied in my laboratory in the medical school. Some common laboratory animals were used in special experiments. The observations on the living corneas were made almost always with a binocular microscope with magnifications ranging from 10 to 216 diameters. Panophthalmitis with keratitis vasculosa was produced in some common laboratory animals by using Arnold's method [6] of injecting red mercuric sulfide (cinnabar), in water, into the vitreous mass.

Except when it was necessary or advisable to inject some of the corneal vessels individually, all of the injections were made from a common carotid artery. India ink was injected rhythmically into this artery until the corneal arteries, capillaries and veins were conspicuously black. In some instances red mercuric sulfide (in water) was injected rhythmically into the same artery until it could be seen in many of the scleral and corneal arteries. Excessive injection of the dye was avoided so that the water would not wash the ink out of the capillaries. The vessels labeled A in figures 5, 6, 9, 13, 14, 15, 16 and 17 are amuscular arteries. Some of these arteries contain a small amount of the mercuric sulfide mixed with ink. Some of the others contain ink only, but they can be unmistakably traced back in the specimens to scleral arteries which contain some of the dye. The vessels labeled *V* in the same figures contain either ink only or ink and blood. These vessels are amuscular veins. The absence of muscle fibers between the tunica intima and the tunica adventitia was demonstrated by using Arnold's methods of staining and microdissection.[6a]

All of the specimens were mounted in dammar after they were cleared, and certain parts of them, such as the uvea, and in some instances parts of the cornea, sclera and conjunctiva, were removed by dissection.

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6. Arnold, J.: Experimentelle Untersuchungen über die Entwicklung der Blutcapillaren: II. Die Entwicklung der Capillaren bei der Keratitus vasculosa, Virchows Arch. f. path. Anat. **54**:1, 1872.

6a. Arnold, J.: Beiträge zu der Entwickelung der Blutcapillaren im embryonalen Glaskörper, Virchows Arch. f. path. Anat. **54**:408, 1872.

The specimens were cleared by the Spalteholz method of dehydrating with alcohol, displacing the alcohol with either benzene or xylene and then displacing the benzene or xylene with methyl salicylate. All of the photomicrographs were taken by using transmitted light.

Some special details concerning the materials and methods are stated in the explanations of the figures and also in the body of the paper.

EVENTS OBSERVED IN CORNEAS BY DIRECT AND INDIRECT MEANS

Vascularization Unaccompanied by Neovasculogenesis.—It is common for the crests of such vascular loops (substitutes for arteriovenous anastomoses) as those shown in figures 1 and 2 to move either steadily or in a jerky manner toward the center of the cornea as the legs of the loops increase in length. The steady movement of the tip of a loop could rarely be observed directly, but it was demonstrated in many corneas by indirect means. For instance, the surface of the cornea was gently pricked with a sharp needle immediately above the tip of a loop, and a small speck of either ink or red mercuric sulfide was tamped into the small wound, so that it was possible to determine whether the tip moved or remained stationary. In some instances the speck of ink or mercuric sulfide was tamped into a small wound at or near the center of the cornea, so that it was possible to measure repeatedly the distances between the speck and the crests of various loops. The distances between the speck and the points of origin of various anastomoses connecting the arterial and venous legs of some loops with one another were also measured systematically in many instances. The measurements showed not only that the crests moved toward the center of the cornea but that either parts or all of some of the cross anastomoses moved in the same direction. Some simple networks of vessels accordingly moved toward the center of the cornea.

The erratic or jerky movements of the vessels are most common during the earlier part of the invasion, as can be explained best by referring to figure 2. I was especially interested in the unusual dilatation of the limbic vessels of this eye and happened to be observing the portion of it shown in figure 2 when a vessel leaped out of the network of limbic vessels and quickly assumed the hairpin-like form, which it still has. The two conspicuous loops shown in the upper right portion of the picture also leaped farther into the cornea as well as somewhat to the left. Immediately before and also during the time that these vessels leaped away from their original positions I saw many small vessels break. The retracted stumps of the broken vessels are shown clearly in the picture. Many of them bled freely from their broken ends. These stumps are the vestiges of some capillaries and other small vessels which should be called restraining vessels, because they temporarily retarded or

restrained certain other vessels in their movements toward the center of the cornea. The cornea was clear until the restraining vessels broke and bled. I have never been able to recall the exact vascular connections of the present hairpin loop with the general network of limbic vessels before the restraining vessels broke, but I saw it assume its present form as it leaped from a relatively posterior position to its present one. The free ends of the vascular stumps attached to this loop were directed sharply toward the limbus for a few seconds after the loop developed. It is remarkable how quickly the open ends of the stumps became sealed so securely that ink did not escape from them. Ink did escape, however, from some similar stumps in certain other parts of this cornea.

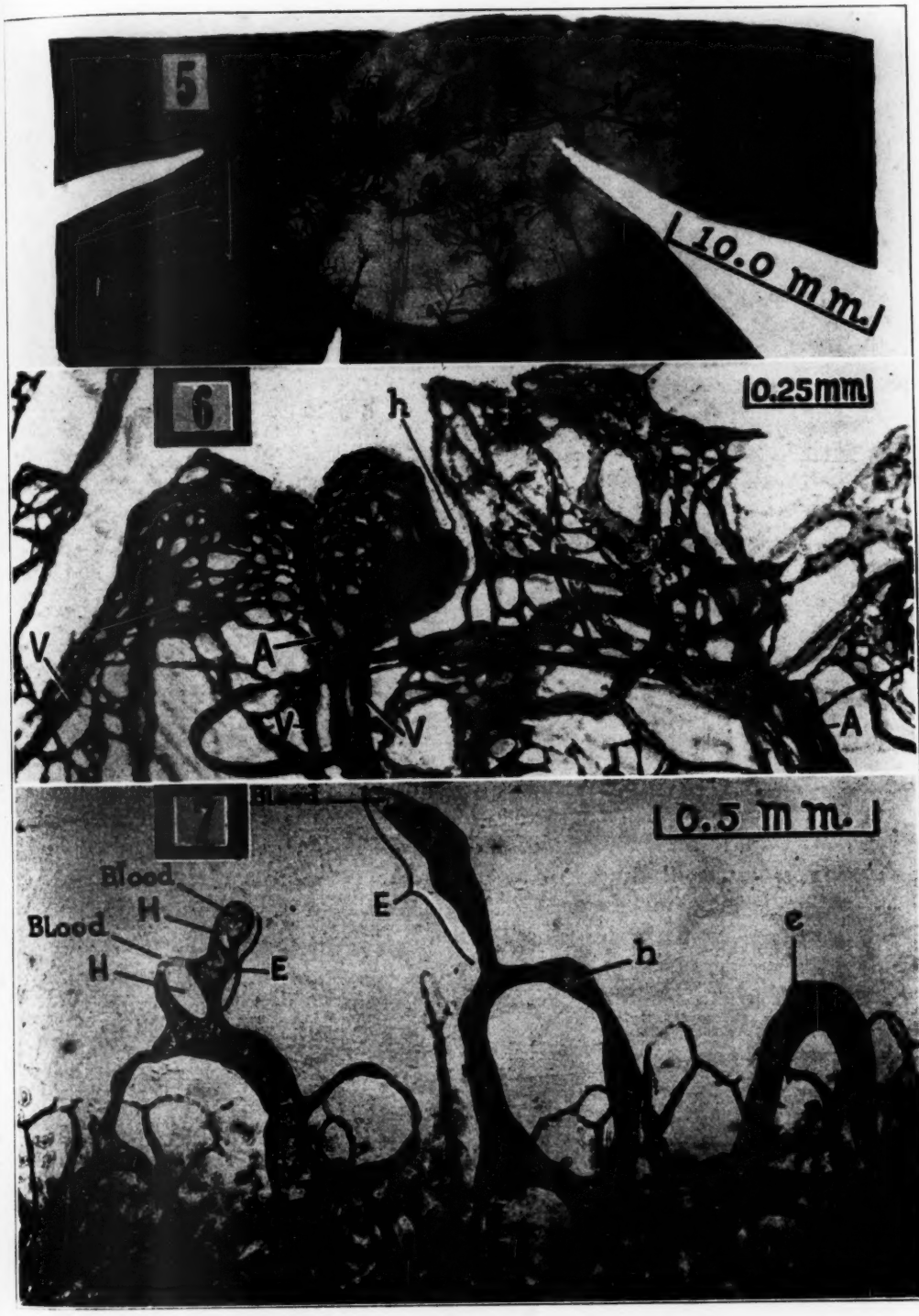
The restraining vessels (fig. 2) broke so rapidly that I am unable to describe accurately the breaking in this instance. In other instances, however, I saw restraining vessels become so slender at or near the middle, as they became longer, that blood ceased to flow through their narrowed lumens shortly before the vessels broke. In some instances the lumen of a restraining vessel became obliterated near both ends, but not at the middle, so that an ampuliform pocket containing either plasma or whole blood existed at or near the middle of the stretched vessel. These pockets rarely contained blood cells. Some of the restraining vessels which I watched persisted much longer than others. In fact, some of them did not break. Their lumens became either obliterated or almost obliterated and then large enough that blood again circulated freely through them. Direct observation showed that at least in some instances the breaking of the restraining vessels was responsible for the jerky or erratic movements of vascular loops and simple networks of vessels toward the center of the cornea. The movements of the substitutes for scleral arteriovenous anastomoses (loops and simple networks) became more gradual as the cornea became depleted of the greater number of the restraining vessels. Many of the stumps of the restraining vessels became filled with static blood and then vanished.

On many occasions I saw straight and relatively straight vessels at and near the limbus become hairpin and arciform loops. The crests of these loops moved toward the center of the cornea as the legs of the loops increased in length. I have watched parts of the relatively large veins and arteries of the circle of Hovius as they moved farther into the cornea after they had moved (unobserved) through the limbus into the relatively clear corneal tissue (fig. 3). I have also observed that the limbic region became almost completely depleted of its relatively small vessels as some of them broke and as others moved far into the cornea (fig. 3). In a few instances I saw small ulcers that developed in the relatively devascularized region near the base of the cornea.

Explanation of Plate

Figs. 5 to 7.—Figures 5 and 6, sections from the eye of an adult black panther which died in uremic coma. The darkest spot in the cornea (fig. 5) is a small foreign body (probably a piece of corroded metal) deeply embedded in tissue. The head of the animal was removed before the blood coagulated in the vessels. The corneal vessels were injected in a rhythmic manner from a common carotid artery, while the other severed vessels in the neck were clamped with hemostats, but the greater part of the ink escaped from the corneal veins while the eye was being enucleated. These veins were then reinjected with ink individually and antidromically from points posterior to the limbus while the vessels were observed with a binocular microscope. At one region of the cornea, the upper right region (fig. 5), some loose ink flowed between two corneal lamellae, but this was later removed by dissection. Parts of the trunks of some injected vessels were inadvertently destroyed while removing the ink. The upper part of the cornea was cut away, and some of the vessels in it were carefully dissected under the microscope in order to examine the tunics and especially the amorphous, jelly-like substance which separates the adventitia from the intima of many corneal vessels. The bouquets of vascular loops are situated at four different levels in the cornea. Figure 6 is a photomicrograph of some capillaries and other small vessels shown within the small elliptic area in figure 5. The ellipse was drawn on the photographic print with pen and ink. The individual vessels in the cornea were not studied while the animal was living. The vessels labeled *A* in figures 5 and 6 are amuscular arteries; those labeled *V* are amuscular veins; and the large vessel labeled *h* in figure 6 is one which assumed its present size and form during an injection with india ink. Many small vessels coalesced to form the large one labeled *h*.

Figure 7, section of an eye from a domestic cat (two-thirds grown). The cause of the keratitis could not be determined. The animal was found by the side of a road. It was so emaciated and weak that it could not stand, but it showed some improvement before it was killed. Six days after it was brought to the laboratory it was anesthetized and killed by cutting the abdominal aorta. This time was chosen to kill the animal because I was ambitious to secure a picture of the vessels in the particular stages of development shown in figure 11. I had observed these vessels carefully for many hours each day for five days. They were injected from a common carotid artery, and the specimen was otherwise prepared as described in connection with figures 1 to 3 inclusive. The structures labeled *E* are diverticula or evaginations which were invading the cornea. Some blood was driven to the ends of these evaginations by the ink. The short structure labeled *e* is a retracted evagination. The clear areas labeled *H* are tissue islands or holes which developed when opposite walls of the evagination met and fused. At the point labeled *h* a small hole or tissue island vanished while the ink was being injected. In this case two small vessels coalesced to form a larger one. The large vessels and also the small ones in figure 7 are epicorneal vessels in the sense that they are on the cornea, although they are between the bulbar conjunctiva and the anterior lamella of the cornea.



Figures 5-7

Vascularization Accompanied by Neovasculogenesis.—The cornea presented as figure 7 was extremely clear. I saw the holes or tissue islands labeled *H* develop and grow larger. I saw a hole or tissue island develop at the point labeled *h,* although this hole disappeared under my eyes while the vessels were being injected with ink. One of the difficult events to observe is the development of such an island in a blood vessel. The only warning that it is about to develop is that the blood at the area is reduced to a thin film. As soon as no blood cells can be seen in the area the island has developed, as can then be demonstrated beyond reasonable doubt by killing the animal and injecting ink mildly into the vessels. It is often the case that the hole is at first very small. In some instances it remains small, but in other instances it becomes larger. I saw similar holes develop in some of the more anterior swamp-like vessels shown in figure 4, in some of the vessels which reached the center of the cornea (fig. 3) and also at and near the tips of some of the loops in the corneas of the lamb (as illustrated at and near the tips of the simple and complex loops in figs. 9, 11 and 18). Every time I observed the development of a tissue island in a simple vessel, in a dilated portion of a vessel or in an evagination from a vessel, I witnessed the genesis of a new blood channel or vessel. In some instances, as in the kangaroo's eye (fig. 4), it seems appropriate to speak of the resulting vessels as swamp channels and of the tissue islands as swamp islands in diminutive blood swamps.

In the cornea of the lamb I watched some evaginations as they assumed different shapes. At times an evagination was long with a sharp tip, and at other times it was shorter and had a relatively blunt tip. I saw some small evaginations vanish by retracting. In some instances the original site of the evagination was marked by only a slight distortion of the wall of the mother vessel. The retraction of such a process can be illustrated by referring to figures 12 and 13. When the photomicrograph presented as figure 12 was taken, the small, sharp process which passed to the right from the lower right hand corner of the upper parallelogram (fig. 12) was somewhat longer than the similar process which passed to the right from the upper right corner of the lower parallelogram, as can be seen in the picture. The photomicrograph presented as figure 13 was taken about six months later from the same mounted specimen. In figure 13 the evagination associated with the upper parallelogram is much shorter than the process associated with the lower parallelogram. The vestige of the evagination labeled *e* in figure 7 is another instance of the partial vanishing of an evagination by retraction.

Vanishing of Corneal Blood Vessels by Migration, by Transformation and by Obliteration.—Blood vessels may vanish from a small or large area by moving out of it into some other part of the cornea. For

instance, as illustrated by figure 3, many vessels vanished from the limbic region by moving toward the center of the cornea. A simple runglike anastomosis connecting the two legs of a vascular loop with one another may be said to vanish partially in the sense that it develops into a simple or complex hairpin or arciform loop with its tip directed toward the center of the cornea. By indirect means already described I have repeatedly observed this kind of vanishing of parts of vessels, entire vessels and even simple networks of vessels from parts of living corneas. Many vessels may vanish from the sclera by moving into the cornea, and they may then vanish from the base of the cornea as they continue to move toward the center of the cornea.

Vanishing of corneal blood vessels by sudden transformation of small vessels into a larger one was observed as a spontaneous event in some inflamed corneas of rabbits. This sudden transformation means only that the holes or tissue islands which have recently developed in a vessel become suddenly eliminated as the mildly fused, opposite walls of the original vessel become separated from one another. As such islands vanish, the small blood vessels diminish in number. In a special sense the small vessels coalesce to form the original one in which the tissue islands had earlier developed. This type of coalescence was successfully brought about in a few instances by clamping the external and internal jugular veins in the neck so that the corneal vessels became so greatly distended with blood that some of the recently fused, opposite walls became passively separated from one another.

Contributory evidence of such a transformation of small vessels into a larger one can be obtained by injecting the substances into the vessels of dead corneas so strongly that some of the recently fused walls are forced apart. For instance, I saw a hole develop at the point labeled *h* in figure 7, and then I saw this hole or island vanish while the vessels were being injected with ink. In another cornea (fig. 6) I saw and studied a network of small vessels in the location of the large vessel labeled *h*. The small vessels were already injected with ink. On injecting more ink I saw many of the tissue islands among these vessels vanish, so that one relatively large vessel existed in the place of the smaller ones. This large vessel is comparable to one shown in the article published by Spicer.[7]

Vanishing of corneal blood vessels as a result of spontaneous obliteration of their lumens is an ordinary event. In the cornea of a Rocky Mountain bighorn lamb a slender stream of blood was circulating, at times rapidly and at times slowly, in the lumen of the arterial leg of a metamorphosing arteriovenous anastomosis which reached from the

7. Spicer, W. T. H.: Parenchymatous Keratitis; Interstitial Keratitis; Uveitis Anterior, Brit. J. Ophth., 1924, supp. 1, p. 29, fig. 30.

Figs. 8 to 18 (inclusive).—Sections from the eyes of a Rocky Mountain bighorn sheep (lamb) which died (probably of starvation) about fifty hours after it was born. The blood vessels in the eyes of this animal were not studied before it died. The injections were made from a common carotid artery (first with India ink and then with a small amount of red mercuric sulfide), and the eyes were further prepared as described in connection with figures 1, 2 and 3.

Figure 9 is a higher magnification of the vessels shown to the right in figure 8. Figure 11 is a higher magnification of the two loops shown in the rectangular area in figure 10; figure 13 is a higher magnification of some vessels shown in figure 12; figure 18 is a higher magnification of the vessels shown in the

(Legend continued on next page)

rectangular area in figure 17. The magnification for figures 10 and 12 is the same as for figure 8 and that for figure 9 and 11 is the same as for figure 13. The magnification is the same for figures 15, 16 and 17 as for figure 14. The letters on the figures have the following meanings: *AVA*, arteriovenous anastomosis; *A*, amuscular artery; *V*, amuscular vein; *VE*, venous evagination.

The arteriovenous anastomoses in a single cornea of the lamb were found to be situated at five different levels in the tissue. The epicorneal plexus of blood vessels was removed from all of the preparations except those shown as figures 10, 14 and 15. Parts of this plexus can also be seen in figure 8. The fine epicorneal meshwork of vessels is not in clear focus in any of the figures. These vessels were developmentally at a standstill. None of the conspicuously sturdy bouquets of vascular loops originated from the epicorneal plexus.

limbus almost to the center of the cornea. Some unilateral irregularities or invaginations of the intima almost obstructed the blood flow at some points. A few minutes later the flow became completely obstructed by at least one of these intimal invaginations. The blood was then static in all parts of the arteriovenous anastomosis. I have not found it possible to reopen the lumen of such a vessel in a dead animal by injecting a liquid into a common carotid artery, although the liquid may enter the venous leg, as shown in the arteriovenous anastomosis to the right in figure 16 and also in the anastomosis to the right in figure 17. Ink was observed to pass antidromically into both of these veins. Incidentally, the venous side of the arteriovenous anastomosis to the right in figure 16 is filled principally with blood. Only the lower part of the main trunk of this venous leg is black with ink. I have failed to find any trace of these bouquets of vascular loops (as in figs. 14, 15, 16 and 17) in the corneas of adult sheep.

It was appropriate to describe the destruction of restraining vessels in connection with the erratic movements of corneal loops. In a general way that description is applicable to the destruction of some loops. This destruction of corneal loops has been described by Julianelle and Bishop.[8] At or near the crest of a loop the vessel becomes first too slender to conduct blood, and then it breaks into two pointed stumps which retract.

According to my observations the tips of one or both of the stumps of a broken loop may be slender and slightly curved for minutes and even hours after the break occurs. The stumps often have the appearance of the "capillary sprouts" which Arnold found in his prepared specimens. Arnold assumed that these "sprouts" arch toward one another, as if by positive chemotaxis, preparatory to fusing to form an arched structure which then becomes an arched "capillary" as soon as "canalization" takes place.[9] Immediately before the break occurs, the arched structure is often identical in appearance with the "capillary sprouts" which Arnold assumed had arched toward one another and fused but had not become completely canalized. The constriction is at times irregular, so that ampulliform blood pockets or plasma pockets exist. Some of the pockets seem to be identical in appearance with pockets which Arnold described but mistook for beginning "canalization" of the "fused" structure at or near the middle, as well as at one or both ends. I observed this destruction of loops in the inflamed corneas of some guinea pigs and rabbits, but I was unable to observe it in the cornea of the lamb. I likewise failed to observe the destruction of restraining vessels in the cornea of the lamb. I was also unable to find any red blood cells free in the cornea of the lamb.

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8. Julianelle, L. A., and Bishop, G. H.: The Formation and Development of Blood Vessels in the Sensitized Cornea, Am. J. Anat. **58**:109, 1936.

9. Arnold, J.: Experimentelle Untersuchungen über die Entwicklung der Blutcapillaren, Virchows Arch. f. path. Anat. **53**:70, 1871; footnotes 6 and 6a.

COMMENT

There are probably several dependent factors which contribute collectively to the looping of scleral vessels into the cornea. One is an appropriate concentration of hydrogen ions in the scleral and corneal tissue to promote rapid proliferation of the cells of the tunics.[1] As this proliferation of the cells takes place, the vessels loop away from their normal positions rapidly if the surrounding tissue is so necrotic (as in certain cases of panophthalmitis) that it offers little resistance to the movement of the vessels and is rapidly eroded by the growing vessels. The partial loss of elasticity of the walls of the vessels in necrotic tissue is a factor of minor importance. Studies of the vascularization of the cornea of the lamb show that the capacity of growing arteriovenous anastomoses to erode the intralamellar scleral and corneal tissue is an important factor contributing to the invasion of the cornea by the scleral vessels. The energy imparted to the vessels by the heart is also an important factor contributing to the looping of scleral arteriovenous anastomoses into the cornea.

If the arterial blood pressure is low, the brunt of the vascularization is often borne by the veins. In this connection I wish to call attention to the inflamed corneas of the kangaroo (fig. 4) and the domestic cat (fig. 7). I am unable to say that an artery invaded the cornea in either of these instances. The arterial blood pressure of the cat was not determined, but it was probably low, at least some of the time, because the heart action was often feeble. The pressure in the left brachial artery of the kangaroo was determined many times with a Tycos sphygmomanometer. The systolic pressure was never found to be higher than 55 or lower than 22 mm. of mercury while the animal was in the comatose state. It is conceivable that the arteries did not move and that the veins moved away from their normal positions because the arterial blood pressure was too low to urge the arteries into the cornea and that the pressure of the blood in the veins was at times great enough to urge these vessels away from their original positions in the sclera. If this reasoning is correct, the venous pressure is in exceptional cases a factor contributing to the invasion of the cornea by veins.

The vascular evagination is an incomplete or imperfect vascular loop. The development of an evagination means that only a local portion of one side of a vessel invades the tissue by moving into it, while the neighboring portions of the vessel either remain stationary or move less rapidly than the tip of the evagination. Local proliferation of cells may promote the incomplete or abortive vascular looping similarly as the proliferation of cells of the tunics of a loop may contribute to movement of the crest of this loop toward the center of the cornea. However, it is not safe to explain the development of the evaginations entirely on the

basis of cell proliferation, because some evaginations become longer and shorter and even vanish by retracting. If proliferation of cells in the walls of an evagination takes place before the retraction occurs, the wall of the mother vessel may have a local thickening due to the presence of an extra number of cells. When such a wall is examined in histologic sections a person might erroneously conclude that he is observing an incipient stage in the development of a vascular "bud" or "sprout" when he is actually looking at a vestige of an evagination.

It is only a relatively small number of the evaginations which meet and fuse with one another to form new blood vessels. Some of them vanish by retracting. Others become distended with static blood and then vanish. The greater number of them divide one or more times so that they become thereby converted into vascular loops as was suspected but not seen by Julianelle and Bishop.[8]

The development of holes or tissue islands is also a result of imperfect or incomplete vascular looping. A local portion of only one side of a vessel moves into the blood stream, while the other parts of the vessel remain either stationary or relatively stationary. The local portion that moves into the lumen of the vessel is an invagination which may touch the opposite wall and fuse with it to form a hole or tissue island, which may be easily destroyed shortly after it has formed. When the intravascular pressure is increased about the newly formed island the mildly fused walls may become separated from one another, and the same island may never develop again. The new vessels which are formed as a result of the fusion of opposite walls of a vessel may or may not form loops which grow in different directions through the tissue.

It is undoubtedly the case that in some instances during life the surrounding tissue may press against a vessel in such a manner that opposite walls of the vessel come passively in contact with one another and fuse, but I am aware of some instances (as in fetal epiphyses) in which the tissue pressure is probably not a factor in the development of the tissue islands. The greater number of the holes or islands which develop in the vessels are probably due to the invaginations of the growing walls of the vessels. Such invaginations are not more remarkable than the diverticula or evaginations which move through the tissue and form new blood vessels and concomitantly new tissue islands by meeting and fusing with one another. It is a common occurrence for at least one invagination to develop in an evagination, as is clearly illustrated in figures 7, 9, 11 and 13. An evagination may become a network of vessels before any part of it meets and fuses with any part of another evagination.

The diverticula shown in figure 7 are essentially the same as the venous diverticulum which forms the upper side of the parallelogram in figure 13. The differences in form are not important, because a single

evagination may assume various forms. The sharpness or bluntness of the tip can scarcely be looked on as being an important feature in any instance because, so far as I have been able to determine, a sharp tip, like a blunt one, has a lumen, although a part of the lumen of a sharp tip may be so small that only plasma and some blood débris can enter it. Such tips often seem to be solid structures when they are seen in prepared specimens. It would be inconsistent to speak of the evaginations as "buds" or "sprouts" without committing the obviously ridiculous act of calling the invaginations "buds" or "sprouts" also.

It is amazing how bald hairpin loops (loops which do not possess evaginations) avoid touching one another in growing through normal tissue, as at and near the center of a highly vascularized cornea of the guinea pig or that of the lamb. A bald hairpin loop may be growing in the direction of another one but either stop growing or merely swerve to one side of the second one in time to avoid touching it. Loops which are provided with or crowned with evaginations behave in exactly the same manner as the bald ones, except that the evaginations of approaching loops may not succeed in avoiding one another and may accordingly fuse to form anastomotic connections between the loops. If the loops consist of capillaries, the haphazard fusion of the evaginations may eventually be responsible for the existence of a complex network of capillaries which connects all of the smallest tips of the arteries and veins of many bouquets of vascular loops with one another. Each of these bouquets should then be spoken of as a completely metamorphosed arteriovenous anastomosis.

The bouquets of vascular loops shown in figures 14, 15, 16 and 17 are incompletely metamorphosed arteriovenous anastomoses. It is true that evaginations are present in these bouquets, but in the living cornea the arteriovenous anastomoses were at so many different levels in the lamellated or stratified corneal tissue, the number of metamorphosing arteriovenous anastomoses at any level was so small and the distribution of these anastomoses at any level was such that the evaginations of any one anastomosis did not have an excellent opportunity to come in contact with and fuse with evaginations of any other anastomosis. It is probable, however, that if some of the metamorphosing arteriovenous anastomoses at a certain level had reached the center of the cornea the metamorphosis would have become complete, as it did in the cornea of the panther (figs. 5 and 6) or as in the eye of the guinea pig presented as figure 3. After the crests of several loops reached the approximate center of the cornea (fig. 3) they avoided one another by twisting and turning in various directions. I then saw (for the first time in this eye) diverticula develop and fuse with one another, and I also saw holes or tissue islands develop in some of the vessels which became distorted

after they reached the center of the cornea. A result of the distortion of the crests of the corneal loops, the development and fusion of evaginations and the development of new blood vessels due to the invaginations is the hemangioma-like tangle of vessels seen at the center of the cornea (fig. 3). Many of the arteries vanished, so that the tangle eventually consisted principally of veins and capillaries. It seems correct to speak of the diverticula or evaginations as fusion evaginations, because, so far as my observations go, vascular loops do not become united with one another if evaginations are not present.

My observations that evaginations rarely fuse with one another in the cornea and that the fusion is haphazard when it occurs (as illustrated in fig. 13) are opposed to the notion of Arnold,[9] Augstein [10] and many others that the tips of "capillary sprouts" regularly approach one another, as if by positive chemotaxis, and then fuse to form straight and arched structures, which soon become "canalized." I am convinced that Arnold's alleged instances of systematic meeting of the tips of his "capillary sprouts" was greatly overestimated owing to his frequently mistaking degenerative events for generative ones while observing his prepared specimens. He undoubtedly mistook stretched portions of restraining vessels and stretched crests of some vanishing loops for fused tips of "capillary sprouts" which had not become completely "canalized," and he undoubtedly mistook the greater number of slightly retracted stumps of vanishing vessels for "capillary sprouts" which were in the act of approaching one another, as if by positive chemotaxis preparatory to the meeting and fusing of their tips. Arnold did recognize a small number of the stumps as being probably the retracted stumps of broken "capillaries." This last interpretation is remarkable in view of the fact that Arnold did not see a "capillary" break.

One of Arnold's findings is important in connection with the phenomenon of the final devascularization of the cornea. This is his observation that each of the greater number of the "capillaries" and even many of the "capillary sprouts" is enclosed in a cylinder of adventitial cells and fibers. He demonstrated this fact by reflecting a part of the adventitial wall to expose the intima (which he considered to be the capillary). In fresh specimens there is a relatively clear space (intramural space) between the walls of the two cylinders of many of the corneal vessels. Some of my microdissections revealed that this space is filled with a jelly-like mass. This is especially evident in the largest arterial trunks. After one of these vessels has existed for a considerable time, the lumen becomes progressively smaller as the intramural space becomes progressively larger. The lumen continues to decrease in

10. Augstein: Gefässstudien an der Hornhaut und Iris, Ztschr. f. Augenh. **8**:317, 1902.

size until it becomes completely obliterated at one or more points along its course. This obliteration can undoubtedly be attributed to such an increase in the amount of the jelly-like mass that the walls of the intima are forced together at one or more points. In other parts of the body the intramural space can be found, but it becomes invaded by muscle and connective tissue and in some instances also by small blood vessels (vasa vasorum).

Some simple loops in the cornea become closed off at or near their crests. This is probably due to a postinflammatory shortening of the legs of the loops, because as soon as a loop breaks at or near its crest the legs sometimes retract to such an extent that the ends of their pointed tips are separated from one another by a considerable distance. In a healing cornea the legs of a loop may be in a relatively healed area near the limbus, while the crest is in a relatively unhealed area and may accordingly be the weakest and most inelastic portion of the loop. If the legs retract, as the surrounding tissue contracts the lumen becomes obliterated at or near the crest before the vessel breaks.

It is certainly incorrect to explain the movements of vascular loops in terms of positive chemotaxis, because the crests of the loops tend to avoid touching one another. I believe that it is equally incorrect to speak of negative chemotaxis as a factor contributing to the tendency of the crests to avoid one another. These crests merely grow best in the avascular parts of the tissue, where the concentration of hydrogen ions is greater than it is in the immediate vicinity of another vessel, where the exchange of respiratory gases and nutrient and waste substances in general is relatively perfect. Loops may accordingly become interdigitated without touching one another. The evaginations are not so successful in avoiding one another, because these blind structures contain static or relatively static blood, so that the exchange of respiratory gases and other substances is not good in the immediate vicinity of these structures. The hydrogen ion concentration is accordingly somewhat high about them, but it is low enough that they show an unmistakable tendency to retract, to stop growing or to swerve gently away from one another when their paths of growth cross or meet. However, some of the evaginations do meet and fuse in some corneas.

SUMMARY AND CONCLUSIONS

An inflamed cornea may become almost completely vascularized by preexisting scleral and conjunctival vessels before a new blood vessel develops in it. In fact, many vessels may become destroyed in the process. This is vascularization unaccompanied by neovasculogenesis. Single and interconnected vascular loops move first erratically and later steadily toward the center of the cornea. The irregular or erratic movement is due to the breaking of some stretched vessels which were called

restraining vessels because they temporarily retard the motion of other vessels in relatively more advanced positions in the cornea. The restrained vessels may leap or rush rapidly toward the center of the cornea every time some of the restraining vessels break. After the cornea becomes depleted of the greater number of the restraining vessels, many parts of the remaining vessels move steadily through the tissue. As the legs of a loop become longer, owing to loss of elasticity of the vessel but principally owing to the proliferation of the cells of the tunics, the crest of the loop slowly erodes the restraining tissue ahead of it and moves farther into the cornea. In some instances the scanty interlamellar tissue is so necrotic that the erosion by the crest of a loop is a negligible factor.

In the apparently normal cornea of the Rocky Mountain bighorn lamb and in some traumatized corneas the crest of a loop may erode the tissue so slowly that at least one incomplete loop may develop and advance beyond the major portion of the crest. This incomplete loop is a diverticulum or evagination with a lumen. It has been erroneously spoken of as an "endothelial bud" or a "capillary sprout." It may have at one time a pointed tip and at another time a blunt one. It may even vanish by retracting, or it may grow longer and meet and fuse with a similar evagination either from the same vessel or from a different one. These incomplete loops should be called fusion diverticula or fusion evaginations, because they are the only vascular structures which were observed to meet in the tissue and fuse with one another. They are excellent tissue invaders, because they are so small and pliable that they can enter and move through extremely small interlamellar and even intralamellar spaces. These diverticula are not excellent fusion structures, because they tend to avoid one another by retracting, by merely ceasing to become longer or by gently swerving away from one another. However, when these fusion evaginations are plentiful some of them may fail to avoid one another and accordingly fuse to form new blood vessels.

Another incomplete vascular loop is the invagination which also originates from only one side of a vessel and increases in length. It does not invade the tissue. It invades the blood stream instead. Its tip touches and fuses with the opposite wall of the same vessel, so that a bloodless area, a hole or a tissue island develops in the vessel and two blood channels or vessels concomitantly develop out of the original vessel. One or more of the tissue islands may develop in the crest of a loop, in a leg of a loop and even in an evagination or diverticulum. Some of the new vessels thereby developed form hairpin or arciform loops. These move into or invade nonvascularized portions of the corneal tissue where they become new sources of development of more evaginations and more invaginations.

Spontaneous destruction of corneal vessels is a common event. For instance, certain vessels (restraining vessels) at and near the limbus become stretched and then broken by some others which are moving relatively rapidly in relatively advanced positions in the cornea. It is also the case that the legs of certain loops retract, so that the crests of these loops become stretched and even broken. This destruction of loops takes place when the crests are in the relatively unhealed portion of the cornea near its center and the legs are in the relatively healed portion near the limbus. As the relatively healed corneal tissue contracts, the legs of some of the loops in this tissue become so short that their crests break. It is also true that the lumens of corneal arteries eventually become small and that such intimal invaginations develop in these arteries that their already narrowed lumens become locally obliterated. If the obliteration takes place in the principal arterial trunk of a bouquet of vascular loops which is not connected by anastomoses with vessels of neighboring bouquets, the entire bouquet (including the principal venous trunk) vanishes. However, if the tips of several bouquets are joined with one another, the greater number of the principal arterial trunks may vanish without bringing immediate destruction to the greater number of the capillaries and veins of the several associated bouquets. The narrowing of the lumens and the development of the intimal invaginations are due to the increase in volume of a jelly-like substance between the intimal and the adventitial cylinders of the amuscular arteries.

ORBITAL HYPEROSTOSIS

ITS OCCURRENCE IN TWO CASES OF MENINGIOMA OF THE SKULL

ARNOLD KNAPP, M.D.
NEW YORK

Thickening of the bony walls of the orbit was formerly classified as hyperostosis circumscripta, as described in Naito's atlas.[1] On the basis of the knowledge of meningiomas following Cushing's [2] investigations, a new interpretation of these bony changes arose and a new classification became necessary. In a recent personal communication Schüller [3] stated:

> In the past ten years, since Naito's book appeared, we have observed some cases where the x-ray diagnosis of osteoma or hyperostosis circumscripta had to be corrected for hyperostosis in meningioma. But there are certain reasons that we cannot abandon the group of circumscribed hyperostosis of the skull; because on one side there are cases without progression which are clinically harmless, and on the other side the circumscribed hyperostosis is the beginning of leontiasis ossea, which gives a bad prognosis and is not operable.

Osteoma of the orbit is quite a different process, as these bony tumors (generally called ivory exostoses) arise on the inner wall of the orbit, usually from the frontal or the ethmoid sinus.

Meningiomas occur between the third and the fifth decade of life, at least a decade later than the pituitary adenomas. They are frequent and occurred in Cushing's series in a ratio of 1 to 4 gliomas.

Cushing has divided meningiomas into two groups; in one the growth is large and more or less irregularly lobulated, with a small stalk of attachment to the dura, and in the other the tumor is termed "en plaque" and is round, flat and slightly elevated and tends to spread over the inner dural surface. The latter type of growth is less frequent and arises usually from the basal meninges. Meningioma is found in the places where the arachnoid villi and their associated cell clusters are situated. The arachnoid villi and the cell clusters act as filters for the cerebrospinal fluid between the subarachnoid spaces and the dural sinuses. They become hypertrophied and are the seats of predilection for the development of meningioma. The capping clusters of endothelial cells

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Read at the Seventy-Fourth Annual Meeting of the American Ophthalmological Society, San Francisco, June 9, 1938.

1. Naito, I.: Die Hyperostosen des Schädels, Vienna, Julius Springer, 1924.
2. Cushing, H.: The Meningiomas (Dural Endotheliomas): Their Source and Favoured Seats of Origin, Brain **45**:282 (Oct.) 1922.
3. Schüller: Personal communication to the author.

frequently show a tendency to the familiar whorl formation, and psammoma bodies are present.

Aoyagi and Kyuno,[4] two Japanese investigators, have studied the usual situation of these cell clusters.

Meningiomas, therefore, have certain definite seats of predilection, and each group, on account of its anatomic seat of origin, possesses a definite characteristic symptomatology. The growths abound along the major sinuses and also in the basal meninges, especially over the tip of the temporal lobe and in the region of the gasserian ganglion. In the latter region the meningioma is of the flat (en plaque) variety and appears to rise from near the pterion, where the meningeal vessels and sinuses so commonly channel the bone.

The overlying bone is involved in about 25 per cent of the cases of meningioma. While this hyperostosis occurs in both varieties, it is particularly prevalent in the flat variety.

It is the following groups of meningiomas with which this paper is concerned: the tumors of the sphenoid ridge and tumors of the sylvian cleft (temporofrontal). In these locations a meningioma is particularly prone to push through the dura and later to invade the overlying bone, causing striking hyperostosis. The bone may be involved in a number of ways. Frequently the bone is enormously thickened, and the canaliculi are filled by tumor cells.

Concerning tumors of the sphenoid ridge, Cushing [5] stated:

> The growths which lie astride the sphenoidal ridge, with a portion of the tumor resting on the orbital plate under the frontal lobe and a portion in the middle fossa indenting the temporal lobe, are fairly common and characteristic. Most of them have been unexpected findings, though one or two have been recognized because of an absorption of the sphenoidal ridge shown by an antero-posterior röntgenogram. They may cause uncinate seizures and not infrequently encroach on the side of the chiasma and produce an homonymous hemianopsia.

Cushing's description of tumors of the sylvian cleft (temporofrontal) follows:

> These are, on the whole, similar to the above and doubtless arise from the same sinus, but they lie sufficiently far lateralwards to be brought into view by a subtemporal decompression. Two of them which gave no localizing symptoms were unexpectedly disclosed in this way and were subsequently removed by an osteoplastic exposure. In this region, too, for some unexplained reason, the tumors *en plaque* are common, and as these meningiomas are apt to provoke hyperostosis and cause a palpable thickening of the bone at the temporo-sphenoidal junction, they are easily recognized through this indication of their presence, as well as by the marked exophthalmos which occurs if the hyperostosis involves the outer orbital wall.

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4. Aoyagi and Kyuno: Neuroglia **11**:1, 1912.

5. Cushing, H.: Cranial Hyperostoses Produced by Meningeal Endotheliomas, Arch. Neurol. & Psychiat. **8**:139 (Aug.) 1922.

The microscopic examination of the tumor tissue shows typical clusters of arachnoid cells arranged in whorls and lying within the dural layers. The bony tumors show bony channels filled with connective tissue and tumor masses.

Meningioma in this location runs a characteristic slow course, with no neurologic symptoms and no increased brain pressure. The principal ocular symptom is exophthalmos, and the eyeground is frequently not changed until late in the disease.

Cushing [5] in 1922 wrote an article on cranial hyperostosis produced by meningeal endothelioma, in which the hyperplasia of the adjacent bone and the possibility that it may overlie a meningeal endothelioma were emphasized. The report showed that this type of tumor may be circumscribed and massive or flat, and if it were not for the associated hyperostosis and its consequences the tumor would not be recognized. In the case reported a smooth hard tumor could be palpated in the left temporal fossa, and the only ocular symptoms were exophthalmos and paresis of the abducens nerve. The roentgenogram revealed increased density in the region of the squamous wing of the temporal bone and of the adjoining part of the sphenoid bone. At operation the temporal muscle was found invaded, and the underlying bone was thickened. This was removed as well as the outer side of the orbit and of the sphenoid wing down to the region of the anterior clinoid processes in the depth of the sylvian groove. At the depth of the operative field the dura was roughened, and when it was opened a flat endothelioma was exposed.

In an excellent article with a good bibliography, Arist Stender [6] described 3 cases in which the meningioma arose from the posterior surface of the greater wing of the sphenoid bone. He also found that meningioma with its origin on the posterior surface of the sphenoid bone frequently presents a characteristic clinical syndrome, with swelling of the temporal and sphenoid bones and extension to the bony orbit, with exophthalmos. The process progresses slowly and gives rise to few symptoms, and there is no increase in the brain pressure. The tumors are usually found in the form of plaques and infiltrate the sphenoid bone and the adjoining bones and occasionally the temporal muscle. Associated with the tumor there is a new formation of bone.

The report of the cases on which this paper is based follows.

B. L., a 44 year old woman, was first seen on Jan. 23, 1922. She stated that the left eye had bulged for one year. There was some headache; vision in the left eye was 20/20, and an exophthalmos of 5 mm. was present. The visual field and interior of the eye were normal; there was weakness of the external rectus muscle. The bone of the outer wall of the orbit was thickened; within the outer

6. Stender, A.: Ueber das Meningiom des Keilbeinrückens, Ztschr. f. d. ges. Neurol. u. Psychiat. **147**:244, 1933.

orbital margin a firm resistance was encountered on palpation, and the whole temporal region was swollen and tender.

The condition was believed to be a circumscribed hyperostosis and an operation was undertaken to remove the excess bone in the orbit. A Krönlein operation was attempted, but the bone was found so hard that an osteoplastic flap could not be prepared, and the bone was removed piecemeal by a chisel and hammer. The postoperative recovery was slow but uneventful. Moderate exophthalmos remained.

On September 15, two years later, the exophthalmos of the left eye was unchanged, but the optic nerve showed neuritic atrophy. Vision was 20/30.

The patient was not seen again until 1924, when the vision in the left eye was found to be lost and the optic nerve was atrophic. Pain about the left orbit had continued.

In 1929 the left eye became more prominent. The bony swelling in the temporal region was greater. The pain about the orbit was still present. The vision in the right eye was normal.

The patient was then lost from observation until 1935, six years later, when the left eyeball was more prominent and the cornea was inflamed from exposure. The eye was removed, and the orbit was found filled with bony masses. After operation there was persistent edema of the lids, and there seemed to be a soft mass in the depth of the orbit. The patient was referred to the Memorial Hospital, and Dr. Hayes E. Martin reported that though a number of roentgenograms were studied no diagnosis was made. On account of the swelling of the lids and edema of the conjunctiva and the patient's pain, an operation was done on December 20; the skin was peeled back from the upper eyelid, and the edematous mass was excised. When the orbital cavity was entered it was found to be filled with a corded, nodular fibrous mass which had the gross appearance and consistence of a hemangioma hypertrophicum. Along the outer wall of the orbital cavity this process seemed to have invaded and destroyed the cortex of the bone or to spring from it. As much as possible of the mass in the orbital cavity was removed. The incision was then extended laterally, and a specimen of bone was removed from the main tumor mass for histologic examination. The incision was closed, and the excess skin of the upper eyelid was pushed back into the orbital cavity and a gauze pack applied. Healing was without incident and the orbital cavity is now entirely healed and covered with skin.

According to Dr. Martin, it was surprising to find that the histologic diagnosis of the material removed from the orbital cavity and also from the bone specimen was meningioma.

The photomicrographs showed meningioma typical in form and structure with new bone formation (figs. 1 to 4).

The roentgen findings in 1935 showed the marked changes in the bones of the skull (fig. 5).

The patient was given roentgen treatments three times a week for a total of fifty treatments. The vision in the right eye remained normal.

The patient was seen on Sept. 8, 1937, when the bony swelling (fig. 6) measured roughly 8 by 6 by 4 cm. The shooting pains were entirely relieved by roentgen treatment.

In this connection it is interesting to note that Velter [7] stated that roentgen treatment of meningioma may have a harmful effect by produc-

7. Velter, E.: Le diagnostic des affections de la région chiasmatique et sellaire, Arch. d'opht. **53**:593 (Aug.) 1936.

ing local anatomic changes (adhesions and vascular changes) which interfere with operation.

The patient in the case reported here presented the characteristic symptoms of unilateral bony deformity of the skull and exophthalmos, with no changes in the optic nerve in the early stage and slight paresis of the external rectus muscle. Roentgen examination showed increased

Fig. 1.—Photomicrograph showing the appearance of the tumor in the dura; low power magnification.

density and enlargement of the squamous portion of the temporal bone and of the large wing of the sphenoid bone, with involvement of the outer and upper walls of the orbit. There is no reason that with the present knowledge the correct diagnosis should not be made at an early stage and the condition relieved by operation when the operative procedure is not formidable.

Elsberg, Hare and Dyke [8] reported a number of cases similar to the one described here and observed 10 meningiomas in 15 tumors associated with unilateral exophthalmos. They found that the protrusion of the eye was due either to perforation of the tumor into the orbit or to thickening of the bony orbital walls or extension of the tumor through the superior orbital fissure and not to pressure on the cavernous sinus or the ophthalmic veins.

Fig. 2.—Photomicrograph showing the appearance of the tumor in the bone; low power magnification.

Cases of this type have been but rarely reported in the ophthalmic literature, though this condition is not unusual and should be recognized by the ophthalmologist to whom the patient is likely to apply because of the striking exophthalmos.

8. Elsberg, C. A.; Hare, C. C., and Dyke, C. G.: Unilateral Exophthalmos in Intracranial Tumors with Special Reference to Its Occurrence in the Meningiomata, Surg., Gynec. & Obst. **55**:681 (Dec.) 1932.

Cohen and Scarff [9] recently reported a case of meningioma of the middle fossa.

The second case was somewhat similar though the condition has shown no progression.

L. E. B., a woman aged 38, was seen on Dec. 13, 1924. Six years before she met with an accident and was thrown against the left side of the head. A roentgenogram showed a bony tumor of the outer wall of the left orbit to be

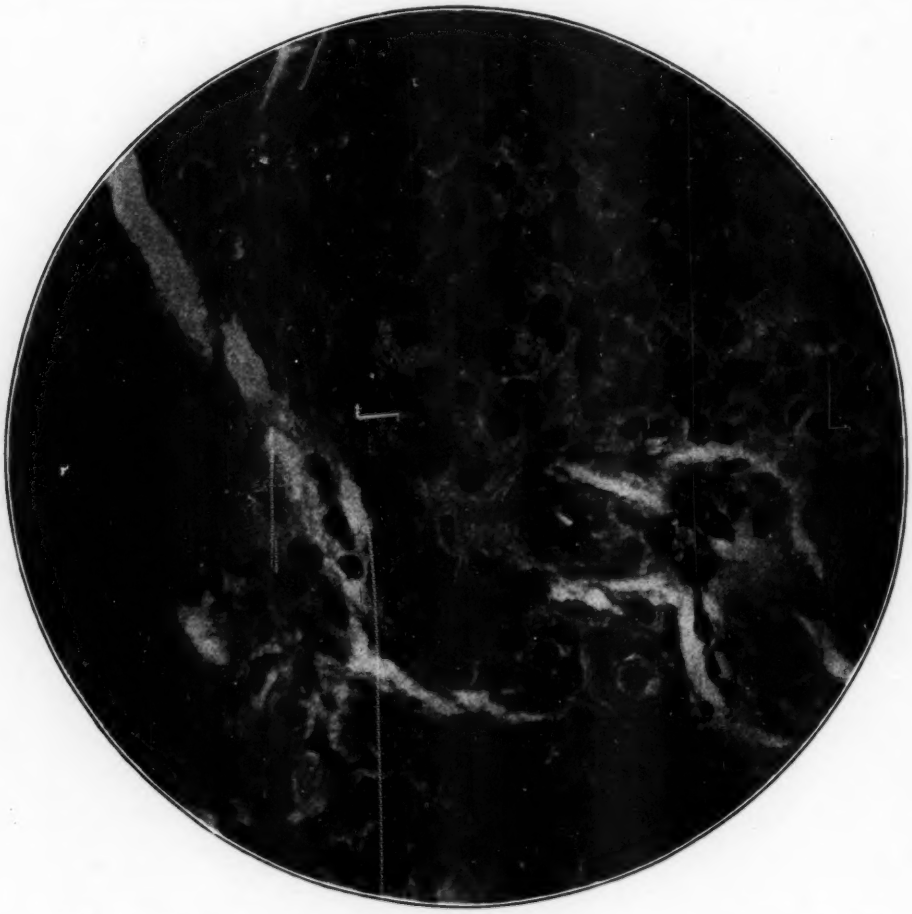

Fig. 3.—Photomicrograph showing the appearance of the tumor in the dura; high power magnification.

present. There has been no change in the past six years. The left eye showed an exophthalmos of 8 mm., and the eye was pushed straight forward. The vision was 20/20—. Motility of the left eye was normal except that the external rectus muscle was weak. There was thickening of the upper outer orbital margin, and a definite tumor could be palpated within the orbit posterior to the external canthus. The optic nerve showed some swelling and neuritic atrophy.

9. Cohen, M., and Scarff, J. E.: Unilateral Exophthalmos Produced by a Meningioma of the Middle Cranial Fossa: Report of a Case, Arch. Ophth. **13**:771 (May) 1935.

The roentgenogram revealed increased density of the bone of part of the left orbit, especially of the greater wing of the sphenoid bone and the outer surface of the frontal bone. The malar bone was slightly involved; the sella turcica was normal. The patient was given roentgen treatment.

In 1936 the condition had not changed. The vision in the left eye was 20/30. The exophthalmos measured 8 mm. The visual field was normal. The optic nerve was pale, and the external rectus muscle remained weak. In reply to a recent inquiry, the patient stated that the condition is unchanged.

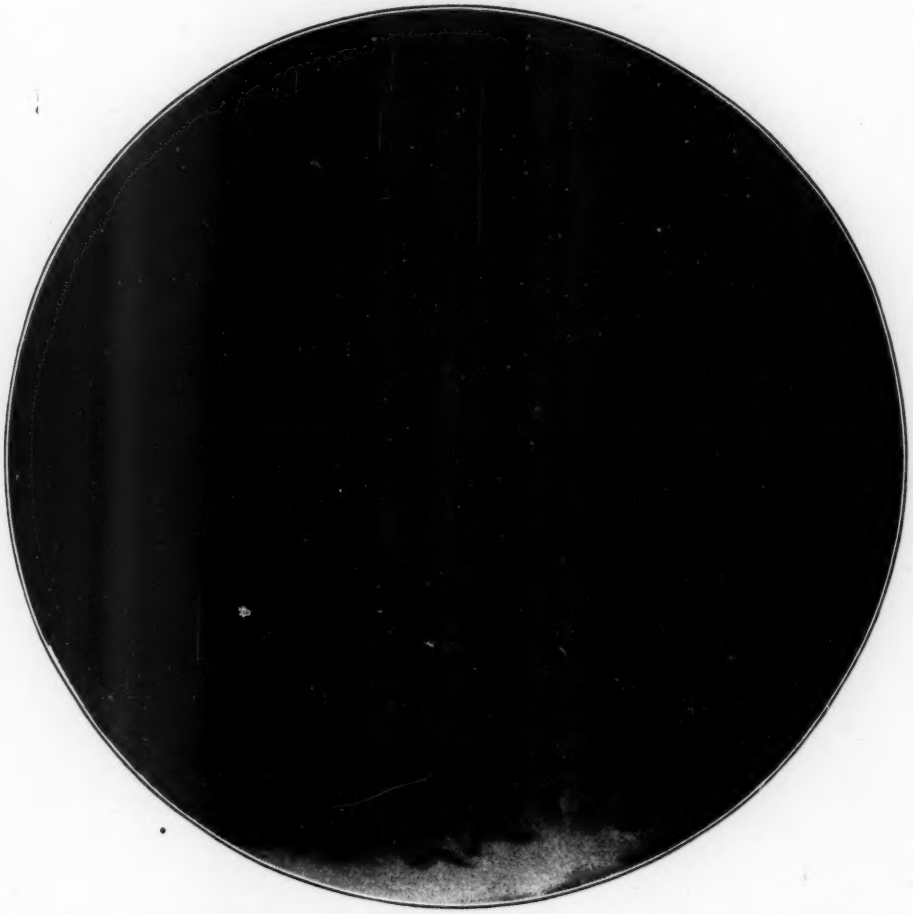

Fig. 4.—Photomicrograph showing the appearance of the tumor in the bone; high power magnification.

There is but little doubt that the meningioma originated at the temporal half of the fissure of Sylvius and involved the greater wing of the sphenoid bone and the temporal part of the lesser wing, extending to the anterolateral part of the frontal, and the anterior part of the temporal, bone. According to Goalwin, in the earlier stages of osseous hyperostosis caused by meningioma characteristic small erosions may be

present on the surface of the hyperostosis, which correspond to the points where the blood vessels enter from the tumor into the bone and are the points where the bony invasion begins.

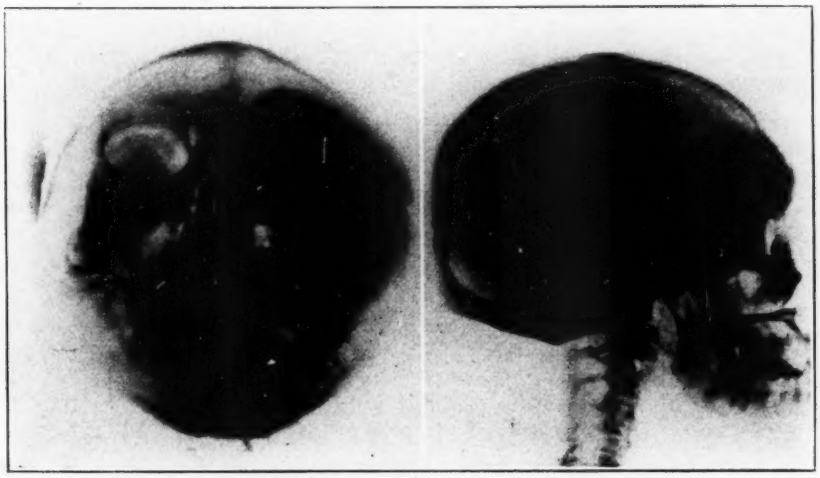

Fig. 5.—Roentgenographic appearance of skull in 1935.

Fig. 6.—Appearance of patient on Sept. 8, 1937.

This case is reported to show that the process may remain stationary; whether this is due to the roentgen treatment or not cannot be determined.

DISCUSSION

DR. W. L. BENEDICT, Rochester, Minn.: (Roentgenograms were shown to demonstrate the three classes of bony tumor of the orbit, the exostoses, enostoses and hyperostoses.) The distinction between these types of tumor is always clear enough to meet the needs of diagnosis and consideration of surgical intervention. The most common cause of orbital hyperostosis is meningioma, but it may occur in cases of primary bone disease due to constitutional dyscrasia or to inflammation or to tumor other than meningioma. When it is believed that vision can be helped or that the condition can be helped by surgical intervention, the method of approach must be carefully considered. Hyperostosis of the sphenoid ridge or of the lesser wing of the sphenoid bone, when accompanied by visual disturbance or by exophthalmos, usually points to meningioma about the sella turcica. Tumors in this position are favorably situated for surgical removal by the transcranial route. Meningioma of the optic nerve in the intraorbital portion has been described. (Roentgenograms and slides showing tumor with hyperostosis of the orbital walls before and after operation were used to illustrate the advantage of the transcranial as against the transorbital approach.)

DR. DANIEL B. KIRBY, New York: I wish to add my experience in a single case in order to stress the matter of early diagnosis, so that the neurosurgeon may operate before the meningioma invades the bones and produces hyperostosis and secondary changes. Hyperostoses are not sensitive to radium or roentgen rays, so the only recourse is surgical treatment. Dr. Knapp pointed out that the meningioma has special sites of origin. Two of these favorite sites are the tuberculum sellae and the sphenoid ridge. My patient was a 65 year old woman. Her first symptom was that of blurred vision in the left eye, which started twenty years ago. There was reason to believe that the changes started in the region of the sphenoid ridge, and rather atypical changes were produced in the visual field. After one year her eye became prominent, and after twenty years it had increased to 32 mm., as measured by the exophthalmometer, and the entire eye was anterior to the lateral orbital margin. The vision and the field of vision in the right eye were normal. All the central vision and the field of vision in the left eye were gone two years after the beginning of the exophthalmos.

Roentgenograms showed an amazing degree of hyperostosis extending through all the walls and invading all the bones, entering into the formation of the orbit. It extended from the roof of the orbit posteriorly and covered the entire floor of the middle fossa; laterally it extended from the region of the tuberculum sellae over the sphenoid ridge out to the lateral wall. The optic canal was obliterated. Only a narrow free space remained in the nasal portion of the orbit. Keratitis developed, and it was necessary to remove the eye. In palpating the orbit one could feel only this narrow space in the nasal portion. Such an extensive bony change cannot be removed surgically. Neurologically, the patient had no objective signs of involvement other than the ocular changes.

The moral in a case like this is that one should exercise more care in obtaining visual fields and roentgenograms in every case of obscure loss of vision and in every case of exophthalmos.

INTRAOCULAR NEMATODE WORMS

REPORT OF A CASE AND REVIEW OF THE LITERATURE

LESTER T. JONES, M.D.

LEWIS W. JORDAN, M.D.

AND

NICHOLAS P. SULLIVAN, M.S.

PORTLAND, ORE.

Many parasites, such as tapeworms, fly larvae and nematodes, are reported in the medical literature as having been found "in the eye." In most instances, however, the parasites have been in the extraocular tissue or conjunctival sac, rather than actually intraocular. Nearly all of the intraocular nematode worms found have been of the genus Filaria and were reported from the tropics.

One of the most interesting cases is that described by Nayar and Pillai,[1] of India.

On June 10, 1932, a native man 34 years of age came to the Government Ophthalmic Hospital at Madras, India, because of blurring of vision and a wormlike object floating about in front of his left eye for five days. Examination of the fundus revealed a "threadlike wriggling worm, coiling itself and moving briskly, especially when light was thrown directly on it. There was an oval clot of blood, about three fourths of the size of the optic disk, near the macula, to which one end of the worm was attached. . . . The worm appeared to be roughly about 3 mm. long. . . ." Fourteen days later the worm disappeared, and after another five days it was found lying in the bottom of the anterior chamber, moving briskly when light was used. Microfilariae found in the blood were of the Filaria bancrofti type. After the worm was removed from the anterior chamber, it was lost.

In 1934 Wright[2] reported the case of a Hindu aged 25, who consulted him for iritis of the right eye.

A fine exudate was seen on the pupillary border, and there were flocculent deposits at the bottom of the anterior chamber with fine keratitis punctata. Fine opacities of the vitreous were present. Details of the retina were not clear. Two round hemorrhages ¼ disk diameter from the macula were seen. Twelve days later a worm was observed moving rapidly in the anterior chamber. A fluorescent sheen gave rise to scintillations as it rapidly lashed and wound itself into a

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Read before the Oregon Academy of Ophthalmology and Otolaryngology in Portland, May 17, 1938.

1. Nayar, K. K., and Pillai, A. K.: A Case of Filariasis Oculi, Brit. J. Ophth. **16**:549-551 (Sept.) 1932.

2. Wright, R. E.: Adult Filaria (Wuchereria) Bancrofti in the Anterior Chamber, Brit. J. Ophth. **18**:646-650 (Nov.) 1934.

loose tangle of coils. It was translucent and faintly opaque. The tail was carried in the position characteristic of the male F. bancrofti, a sharp complete loop being maintained just in front of the anus. Punctate brownish deposits were seen all over the endothelium, and there were fine dustlike floaters in the aqueous. The worm was removed through a small keratome incision. The parasite was 22 mm. in length and 0.09 mm. in width, and it tapered toward the anterior end, which was slightly enlarged and bulbous. The posterior end was characteristically coiled. The worm was identified as F. bancrofti.

Fernando [3] reported from Africa in 1934 the removal of an adult female of the species F. bancrofti from the anterior chamber of the eye. No microfilarias were found in the blood of this patient, who was not from an area in which Filaria was endemic.

F. bancrofti [4] normally inhabits the lymphatics of the pelvis and lower part of the abdomen, where it causes lymphatic obstruction. Here it gives birth to living microfilarias, which are about 200 microns long and are present in the peripheral blood, usually only at night. Some of the microfilarias are taken up by the Culex fatigans mosquito, which acts as the most important vector. However, many different types of mosquito may transmit the disease. After a definite stage of development in the mosquito, the larvae are injected into a new host by a bite of the insect. In the new host the larvae quickly find their way to the lymphatics, where they develop into adults. After about twelve months microfilarias appear in the peripheral blood of the host. Typically, the patients may show lymphangitis of the involved lymphatics, which is followed later by the symptoms of chronic lymphatic obstruction, such as elephantiasis. However, the symptoms of lymphatic obstruction are usually not present in temperate climates. Occasionally the young larvae find their way into the blood stream, thus accounting for their presence in many different regions of the body.

The term Filaria oculi humani [5] or Agamofilaria oculi Stiles [6] has been applied to the organisms in a group of reported cases in which the worms were lost or were not definitely classified and identified after their removal from eyes. One of the earliest cases on record was reported by Mercier in 1771; the involvement occurred in the eye of a Negress. Barkan,[7] of San Francisco, in 1876 reported the case of a patient from

3. Fernando, S. E.: Ocular Filariasis (Adult Wuchereria Bancrofti in the Anterior Chamber of the Human Eye), J. Trop. Med. & Hyg. **38**:17-18 (Jan. 15) 1934.

4. Craig, C. F., and Faust, E. C.: Clinical Parasitology, Philadelphia, Lea & Febiger, 1937.

5. Elliot, R. H.: Tropical Ophthalmology, New York, Oxford University Press, 1920, p. 173.

6. Wood, C. A.: The American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1918, p. 9316.

7. Barkan, A.: A Case of Filaria Medinensis in the Anterior Chamber, Arch. Ophth. & Otol. **5**:151, 1876.

Australia who had a threadlike worm attached to the lower part of the iris. After removal, the worm was thought by Knapp to be Filaria medinensis. However, it is now believed that this worm was probably an unknown Australian species. López,[8] in Havana, saw an active, white threadlike worm 25 mm. in length in the anterior chamber of the eye of a European woman 61 years of age. It had caused slight keratitis and iritis. The parasite passed into the posterior chamber, where it died. Permission for removal was refused by the patient. Von Nordmann[9] found two dead filarias, which were threadlike and 2 mm. in length, in a lens sent him by von Graefe. Gescheidt[10] discovered three filarias, varying in length from 1.5 to 4.5 mm., in a cataract sent him by von Ammon, of Dresden. Schöler[11] in 1875 showed the members of the Medical Society of Berlin a living worm in a human lens. Virchow pronounced it a nematode; it was from 12 to 15 mm. long and moved continuously.

REPORT OF A CASE

On Nov. 12, 1937, H. F., a widow aged 42, was seen complaining of pain in her right eye and epiphora. There was mild iritis; the lacrimal ducts were obstructed, and a large amount of fluid pus was expressed from both tonsils. Atropine was instilled into the conjunctival sac. Salicylates were used at home, and the iritis cleared in seven days. Vision was 20/15 in each eye. A diagnosis of iritis, probably due to tonsillar sepsis, was made.

On April 7, 1938, the patient stated that she woke up with some redness in the right eye, but no pain. At noon she had a sudden sharp pain in the eye. It was an agonizing pain, stabbing in character, and kept recurring at intervals of five minutes. She had severe headache. The redness and swelling of the eye increased. The patient consulted us that afternoon. We found circumcorneal injection, iritis and a contracted pupil. The fundus was normal. A large amount of pus was again expressed from the tonsils.

A 2 per cent solution of homatropine hydrobromide was instilled, and the patient was advised to use hot packs and to take salicylates. The diagnosis was iritis, probably due to tonsillar sepsis.

On April 8 the pain was still severe. The pupil had again contracted. A 1 per cent solution of atropine was instilled at the office. After the patient waited one-half hour, the lower third of the iris was still adherent to the lens. Epinephrine in a dilution of 1:100 was applied to the limbus, and the iris was retracted from the lens without evidence of pigment deposits. The application of a 1 per cent solution of atropine was prescribed three times a day.

On April 11 the eye was greatly improved.

On April 13 the patient felt worse again. The tension was found to be 55 mm. (Schiötz). The use of the atropine was discontinued. Physostigmine salicylate was instilled at intervals of five minutes, until the pupil contracted moderately.

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8. López, E.: Filaria en la cámara anterior, Rev. de cien. méd. **6**:269 (Dec. 5) 1891.

9. von Nordmann, A.: Mikrographische Beiträge zur Naturgeschichte der wirbellosen Tiere, Berlin, G. Reimer, 1832.

10. Gescheidt: Die Entozoen des Auges: Eine naturhistorische, ophthalmonosologische Skizze, Ztschr. f. d. Ophth. **3**:405, 1833.

11. Schöler: Berl. klin. Wchnschr. **12**:682, 1875; **13**:8, 1876.

On April 14 the tension was still increased. Dr. Clinton Cooke was called in consultation and discovered a worm in the anterior chamber.

On April 18 the patient returned to our office. An adult type of nematode worm was easily seen in the anterior chamber of the right eye. Moving pictures of its rapid coiling movements were obtained. The visible part was about 7 to 9 mm. in length and 0.1 to 0.2 mm. in diameter. It seemed to emerge from an oval pocket at the base of the iris, in which it was attached. Like F. bancrofti of Wright, it seemed to taper toward its free end and to throw itself into a "loose tangle of coils," especially when strong light was thrown on it. The patient was referred to Dr. Merle Taylor, who gave the following report:

"The anterior chamber is of normal depth. There is a slight increase in cell content in the aqueous. Six keratitic precipitates are seen on the posterior surface of the cornea at the lower border of the pupillary area. The iris is clear, except at the point of attachment of the worm."

Appearance of the eye with a living adult form of nematode attached to the iris.

No parasites or ova were found in the stool on repeated examinations. No microfilarias were found in smears of the centrifuged blood taken during either the day or the night. Eosinophilia was not present.

On April 23 the worm was removed by Dr. C. Cook. As the worm was lost, it was impossible to tell its actual length, type, etc.

This patient had never been in the tropics. She had lived in Los Angeles from 1927 to 1933, in Sacramento ten years prior to that and in Portland for the last five years. She had a severe attack of herpes oculi in 1933, which lasted for two months. Two years before the present complaint she was bitten by a tick at Wind River, Wash. This bite was swollen and painful for six days. Six months later, while on a hunting trip at Wind River, she was severely bitten by mosquitoes and had a swollen face for several days.

COMMENT

For the present, at least, this parasite must be classed as one of the so-called Agamofilariae. We might assume that it was F. bancrofti. This is supported by several facts. Our worm seems to conform in diameter and gross morphologic structure with this type. The most

common vector of F. bancrofti, the Culex fatigans mosquito, is present in the vicinity in which our patient was living. It is possible that many persons with microfilarias of the F. bancrofti type in their blood have entered such a seaport city as Portland and have acted as sources of infestation. However, our patient showed no eosinophilia and no microfilarias in the blood and had no symptoms aside from those referred to the eye. The adult specimen of F. bancrofti is usually described as being at least 22 mm. in length.[12] The small size of our worm might be explained by its not being removed completely or by the fact that filarias removed from the eye are usually small and immature and represent a youthful migratory stage. The infestation may have occurred several months before, and the first attack of iritis may have marked the implantation of the small blood-borne larva in the ciliary body or iris.

DIFFERENTIAL DIAGNOSIS

Because of the smooth cuticle and the absence of segmentation, we can designate the worm removed from our patient as a nematode. In addition to F. bancrofti, several other possible species must be considered. Filaria loa, or the "eye worm," can probably be eliminated by the absence of its vector, Chrysops dimidiatus (mango fly), from the regions in which the patient was living. In addition, our patient had no Calabar swellings, which are characteristic of this condition. Ascaris lumbricoides must be considered, because at one stage of its development its larvae are present in the blood stream. However, the ascaris even in its early forms is a broad and heavy worm, while our worm had a thin, threadlike structure. Oxyuris vermicularis (pinworm) and Trichuris trichiura (whipworm) pass their entire life cycle in the gastrointestinal tract and never at any time enter the blood stream or lymphatics. They can therefore both be ruled out. The adult form of Trichinella spiralis (Trichina) is never longer than about 3 mm. The fact that the visible part of our worm was at least 9 mm. long completely excludes this possibility. Ancylostoma duodenale and Necator americanus (hookworms) are rather stiff worms and do not show the rapid coiling movements manifested by our worm. We have been unable to find a report in the literature of an intraocular adult hookworm. This parasite can probably further be eliminated by the total absence of clinical findings and ova in the stool.

Outside of the nematode group, the tapeworms Taenia solium and Taenia echinococcus may be considered. However, when these worms occur in the eye they are in a larval cystic form with a short protruding

12. Wright, R. E.; Iyer, P. V. S., and Pandit, C. G.: Description of an Adult Filaria (Male) Removed from the Anterior Chamber of the Eye of Man, Indian J. M. Research **23**:199-203 (July) 1935.

neck. This in no way resembles the worm under consideration. Fly larvae have been found within the eyeball many times. They always have a spindle-shaped segmented form, which in no way compares with a coiling nematode. A Schistosoma blood fluke can be ruled out because it is a flat worm, while ours was a round one. In addition, it has never been reported as found in the eye.

That this worm was a parasite normally infesting animals in the northwestern states and appearing in man as an erratic is a possibility. Filarias have been found in the ventricles of hearts of dogs in the Southern states. Dr. R. Parker, of the Rocky Mountain Laboratory of the Public Health Service in Hamilton, Mont., in a personal communication, made the following statement:

> A number of wild and domestic animals in the United States are infested with Filaria. This group includes deer, porcupines, moose, horses, cattle, and jack rabbits. Worms have been found in the eyes of moose and horses. Little is known of their insect vectors or of the ability of these filariae to infest man.

Many other Filariae which infest human hosts have been described. Most of these were found in the tropics. However, none of them has been observed within the eye.

OTHER CASE REPORTS IN THE LITERATURE

Bacheler,[13] of Gabon, Africa, in 1880 reported the removal of a worm of the species F. loa from the eye of a native young man. It was on the iris beneath the sclera. In 1894 Coppez [14] reported the removal of a dead worm of the same species, 15.2 mm. in length, from the eye of an infant Negress who came from the Congo. It was rather incompletely identified. Filaria equina is often found in the abdominal cavity of the horse in India. Drake [15] reported removing a worm of this type from the eye of a young woman in Madras, India. Since that time, the identity of this particular specimen has been doubted.

Calhoun,[16] of Atlanta, Ga., in 1937 found an Ascaris lumbricoides larva in the anterior chamber of the eye of a boy. No ova were found in the stools, and there was no eosinophilia. In the same year, Tso-Chen-Te [17] described a specimen of Cysticercus cellulosae attached

13. Bacheler, H. M.: The Eye-Parasite, Dracunculus Loa, M. Rec., New York **17**:244, 1880.

14. Coppez, H.: Un cas de filaire dans la chambre antérieure d'un oeil humain, Arch. d'opht. **14**:557-562, 1894.

15. Drake, B.: Ophth. Rev., London **13**:331, 1894.

16. Calhoun, F. P.: Infra-Ocular Invasion by the Larva of the Ascaris, Arch. Ophth. **18**:963-970 (Dec.) 1937.

17. Tso, C. T.: Intraocular Cysticercosis, Chinese M. J. **51**:545-548 (April) 1937.

to the retina. There were no ova in the stools. In 1933 Wilson [18] reported a small thread worm of the Onchocerca volvulus type attached to the macula.

SUMMARY

A case is reported in which a living adult form of nematode, measuring about 9 mm. in length and 0.2 mm. in diameter, was found attached to the iris of a patient who had never been in a tropical country. This parasite was classified as an Agamofilaria because it was lost after its removal and because there were no other physical or laboratory findings to assist in its identification. The differential diagnosis is presented, and cases of intraocular parasites reported in the literature are reviewed.

18. Wilson, R. P.: Onchocerciasis of the Macula, in Eighth Annual Report of the Giza Memorial Ophthalmic Laboratory, Cairo, Schindler's Press, 1933, pp. 85-87.

SCLEROMALACIA PERFORANS

REPORT OF A CASE IN WHICH THE EYE WAS EXAMINED MICROSCOPICALLY

F. H. VERHOEFF, M.D.
AND
MERRILL J. KING, M.D.
BOSTON

Van der Hoeve [1] in 1930 described before the Dutch Ophthalmological Society 2 cases of an ocular condition which he regarded as a clinical entity and designated scleromalacia perforans. In 1932 Rochat [2] described before the same society 2 similar cases under the designation scleritis necroticans. In 1934 van der Hoeve [3] reported in somewhat more detail his own 2 cases and those of Rochat. Including these cases, we have been able to find in the literature references to only 14 cases of this ocular condition.[4] The inclusion of 1 of these cases was doubtful. The patient was aged only 34; there was no arthritis, and the only lesion consisted, even when the patient was first observed, of a filtering cicatrix at the corneal limbus.[5] Three cases, mentioned in the discussion on Rochat's communication, were described simply as being closely similar to his cases, with the exception that in 1 there was no arthritis. We have analyzed the data obtainable from the reports of these 14 cases.

From the Howe Laboratory of Ophthalmology, Harvard University, and the Massachusetts Eye and Ear Infirmary.

Read before the Section on Ophthalmology at the Eighty-Ninth Annual Session of the American Medical Association, San Francisco, June 15, 1938.

1. van der Hoeve, J.: Scleromalacia perforans, Nederl. tijdschr. v. geneesk. **2**:4733, 1931; abstracted, Zentralbl. f. d. ges. Ophth. **26**:504, 1932.

2. Rochat, G. F.: Scleritis necroticans, Nederl. tijdschr. v. geneesk. **77**:1935, 1933.

3. van der Hoeve, J.: Scleromalacia Perforans, Arch. Ophth. **11**:111 (Jan.) 1934.

4. These cases are referred to specifically in the text with the exception of that reported by C. T. Eber (Fistula at Limbus [Scleromalacia Perforans], Am. J. Ophth. **17**:921, 1934).

5. Arkle, J. S., and Ingram, H. V.: ? Scleromalacia Perforans, Tr. Ophth. Soc. U. Kingdom **55**:552, 1935.

ANALYSIS OF FOURTEEN RECORDED CASES OF SCLEROMALACIA PERFORANS

Age.—Of 9 patients, the youngest was 54 and the oldest 76. The patient in the doubtful case was aged 34. The age of 2 of the 4 other patients was not given, and 2 were said to be "old."

Sex.—Seven of the patients were females and 5 were males. The sex of 2 was not stated.

Eyes Affected.—Both eyes were affected in 8 cases and 1 eye in 3 (including the doubtful case). It was not stated which eye was affected in 3 cases.

Situation of Scleral Lesions.—The lesion was situated between the corneal limbus and the equator in 8 cases and at the limbus only in 3 (including the doubtful case). The location of the lesion in 3 cases was not stated.

Character of Scleral Lesions.—The scleral lesion was not definitely described in 3 cases. There were only scleral cavities in 8 cases (including filtering cicatrices at the limbus in 3), nodules and cavities in 3 cases (nodules described as abscesses in 2) and "sequestrums" in cavities in 2 cases.

Period Between First and Last Observation.—There was a period of one year between the first and last observation in 1 case, of eight months in 1 case and of four months in 1 case. The intervals between examinations in 11 cases were not definitely stated.

Recurrences.—There were no definite data as to recurrence.

Ocular Complications.—There were no central corneal opacities in any case. Descemetitis was present in 1 case, posterior synechia in 2 cases, atrophy of the iris with holes in the iris in 1 case, cataract in 3 cases (in only 1 was it secondary to the scleritis); marked opacities of the vitreous in 1 case, staphyloma in 1 (ruptured) and purulent infection in 1 (both eyes). The presence of glaucoma was not mentioned. The fundus was normal in 1 case, and its appearance in the remaining 13 cases was not mentioned.

Visual Acuity.—The visual acuity was normal or was only slightly affected in 4 cases (including the doubtful case) and was markedly reduced in 6. The status of the visual acuity in 4 cases was not given.

Biopsy of Active Lesions.—Biopsy was done in 2 cases, "granulation tissue" and "chronic inflammatory tissue" being reported.

Bacteriology.—Cultures were made in 2 cases (negative), and animal inoculations were carried out in 1 case (negative).

Association with Rheumatoid Arthritis.—Arthritis was present in 10 cases and absent in 4 (including the doubtful case). The duration of

the arthritis before the onset of scleritis was ten years in 1 case, twenty-one years in another case, "chronic" in 2 cases and not reported in 10 cases.

In 1 case [6] there were scleroderma and porphyrinuria in addition to rheumatoid arthritis. (It seems unlikely that either of these conditions were of etiologic significance in regard to the ocular disease.)

From the reports of these recorded cases we have composed the following clinical description of the disease: In one or both eyes there develop simultaneously or at intervals slightly elevated nodules involving the sclera and overlying tissue and situated anywhere between the corneal limbus and the equator. Congestion is moderate and is limited to the nodules and their immediate vicinities. After a considerable period, in some cases six months or longer, one or more of the nodules may disappear. At the former site of a nodule there is seen in the sclera a shallow cavity, often of large size. Over a cavity the conjunctiva may be intact or absent. When, as rarely happens, a lesion is situated at the corneal limbus, it may perforate into the anterior chamber and produce a cystoid nodule such as occurs after trephining. Into this nodule the iris may or may not be herniated. When situated elsewhere, the lesions seldom if ever perforate the sclera, and the remaining tissue usually withstands the intraocular pressure, rupturing only in rare instances. Evidences of intraocular inflammation are relatively slight or entirely lacking, unless, as rarely happens, purulent infection occurs at the site of a cavity. Pain may be absent. Posterior synechiae, atrophy of the iris or cataract may occur as late complications in cases of marked involvement. Usually the ocular disease does not occur before the patient reaches the age of 50. In the great majority of cases definite evidences of rheumatoid arthritis exist at the outset of the scleritis.

We have had opportunity to study clinically, bacteriologically and histologically the following case of scleromalacia perforans, the first in which an entire eye affected with this disease has been obtained for microscopic examination.[7]

REPORT OF CASE

History.—C. M., a married white man aged 52, was admitted to the Massachusetts Eye and Ear Infirmary on Dec. 9, 1936, complaining of a "sore tumor" on the left eye, poor vision in this eye and headaches.

He recalled that his left eye became red about one year before and was certain that the redness was localized in the outer portion of the eye. The eye became "sore," and the redness increased somewhat. He had noticed the "tumor" for at

6. Kiehle, F. A.: Scleromalacia: Report of a Case, Am. J. Ophth. **20**:565, 1937.

7. The bacteriologic investigations were made by Dr. King. The histologic investigations and the deductions from them were made by Dr. Verhoeff.

least three months. The vision had been poor in the eye for several years. For a few weeks before admission to the infirmary he had had frequent attacks of severe headache, with pain radiating down the back of the neck. There was no history of trauma.

About fifteen years before, the patient first noticed that he had some soreness of the feet—they were tired and ached after he had been standing for only a moderate length of time. About fourteen years before, he had his appendix removed, and about eleven years before he was seriously ill for four months with "inflammation of the bowels." At about that time he began to have pain and swelling in the joints. These began in the ankles and gradually affected practically every joint in his body. He continued to work, however, until seven years before the present attack, at which time pleurisy developed. He was then hospitalized for a period of about three months and was discharged from the hospital with a final diagnosis of rheumatic fever and pleurisy with effusion. All examinations in that hospital as well as at a tuberculosis sanatorium had failed to show evidence of a tuberculous origin for the pleurisy. Since this acute rheumatic episode he had been compelled to use crutches or a wheel chair, and his arthritic condition had remained about stationary.

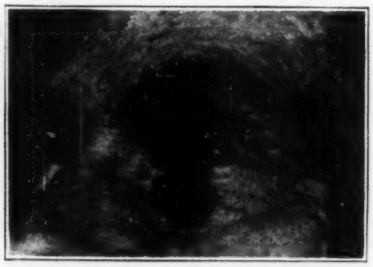

Fig. 1.—Appearance of the left eye when the patient was first seen on Dec. 9, 1936. One lower nodule is hidden by the lower lid.

The family history was essentially unimportant.

Ocular Examination.—The vision of the right eye was 20/20, and the eye was normal. The vision of the left eye was 6/200 with correction. The cornea was clear; the pupil was round and reacted normally. The media were clear. The tension was normal. Situated on the sclera, at the outer side of the eye, were five smooth, waxy-appearing nodules (fig. 1). The largest of these, about 5 mm. in diameter and elevated about 2.5 mm., was situated slightly below the horizontal meridian, with the anterior border about 2.5 mm. from the corneal limbus. The other nodules, each about 3 or 4 mm. in diameter, were situated close to the largest nodule, two being above and two below it. The nodules were covered by conjunctiva and were immobile, firm and tender to pressure. The conjunctival vessels around the nodules were considerably distended. Clinically, the picture somewhat suggested tuberculous nodular scleritis.

Examination of the Fundus.—The optic disk was normal. In the macular region there were fine changes which suggested cystoid degeneration. The fovea was poorly defined. The retinal vessels showed no definite sclerosis. There were a few drusen. In the upper temporal region there were several small pigmented spots and one fairly large irregularly pigmented area of healed chorioretinitis.

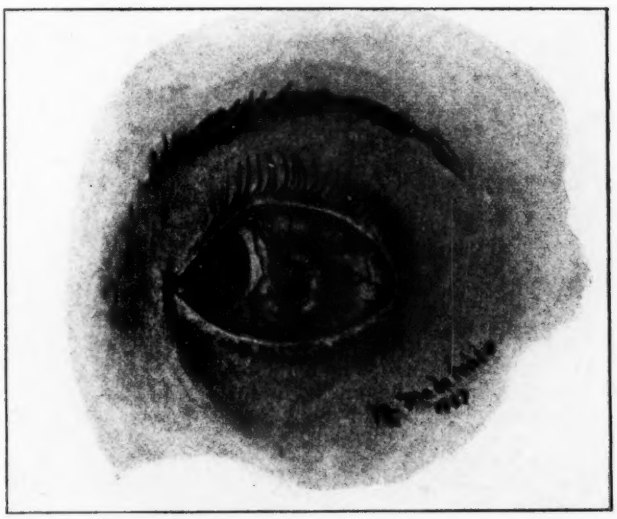

Fig. 2.—Appearance of the left eye just before its removal four months later.

Physical Examination.—General physical examination made by members of the staff of the Massachusetts General Hospital revealed the presence of rheumatoid arthritis, with involvement of the hands, wrists, elbows, shoulders, feet, ankles, knees and hips and the cervical portion of the spine. Old fibrous pleurisy was the only other important finding.

Laboratory Examinations.—The Wassermann reactions of the blood and spinal fluid were negative. The gonococcus fixation test gave negative results. The sputum did not show any tubercle bacilli. The urine was normal. The blood contained 90 per cent hemoglobin, 14,500 white blood cells and 80 per cent polymorphonuclears. Chemical examination of the blood showed 93 mg. of sugar per hundred cubic centimeters, 23 mg. of nonprotein nitrogen and 3.16 mg. of uric acid (plasma). The blood pressure was 140 systolic and 92 diastolic. The reaction to the Mantoux test with 0.1 mg. of tuberculin was negative, while that with 1 mg. was positive The prostatic secretion did not contain any white blood cells or cocci. The electrocardiograms were within normal limits. Roentgen examinations revealed a picture consistent with that of rheumatoid arthritis. There were no calcified nodes in the chest, neck or abdomen. The apical pleurae were thickened.

One week after the patient's admission to the infirmary it was decided to remove a bit of the largest nodule for histologic and bacteriologic studies. The conjunctiva was dissected from this nodule, and with a sharp knife a horizontal incision was made into the nodule just below its midpoint. Immediately on withdrawal of the knife about 3 drops of thick yellowish material escaped from the incision. This material was collected, and a small piece of the wall of the nodule was excised. The conjunctiva was then carefully sutured at the site of the incision. The conjunctiva healed quickly, and the patient was discharged to his home.

Laboratory Examinations of Biopsy Material.—Smears made of the purulent material which was obtained at the time of performing the biopsy were stained with Giemsa's and Wright's stains. Only pus cells and occasional plasma cells were identified in these smears.

Other smears were stained by the Gram method and with methylene blue, dilute fuchsin and the Ziehl-Neelsen stain. In none of these smears were bacteria or fungi found.

The remainder of the purulent material together with a portion of the excised tissue was ground in a mortar. Some of this mixture was transferred to various kinds of bacteriologic mediums for cultural studies. The remainder was then injected beneath the conjunctiva of a rabbit's eye and into the groin of a guinea pig.

A portion of the excised tissue was fixed in Zenker's solution for histologic examination.

All attempts to cultivate bacteria and fungi aerobically, as well as under partially increased carbon dioxide tension, proved fruitless. The results of the animal inoculations were also negative. The guinea pig was found to be normal when it was killed two months after the injection. In the rabbit's eye, beneath the conjunctiva at the site of the injection, a slight reaction developed, but this permanently disappeared within four days.

Microscopic Examination of the Excised Tissue.—The specimen consisted of episcleral and scleral tissue greatly infiltrated with lymphocytes and plasma cells but entirely free from purulent infiltration. Adherent to it was a small mass of pus cells, among which were a considerable number of eosinophils—about ten in a high power field. Presumably this mass of cells was part of the secretion from within the nodule.

Subsequent History.—Eight weeks after the biopsy was done the patient returned to the clinic for admission to the arthritis service at the Massachusetts General Hospital. He stated that the "tumor" had since gradually decreased in size, but that the ocular pain had persisted. The right eye still remained normal, with vision of 20/20. The left eye was considerably injected in the temporal half. Its visual acuity was still 6/200. The largest nodule had entirely disappeared, and in its place there was an almost circular depressed area or cavity in the sclera about 4.5 mm. in diameter. The area had a dark bluish appearance, and its anterior margin was 3.5 mm. from the corneal limbus. Around it the other nodules remained. These nodules were still smooth and waxy and were covered by conjunctiva and elevated about 2 mm. The tension of the eye was normal, and the cornea was clear. The pupil reacted to light and in accommodation. The media were clear, and the fundus picture had not changed.

Progress in Arthritis Ward.—The patient was placed on an extensive regimen in the hope that the advance of his arthritis might be stopped and some of his deformities corrected. A subcutaneous nodule on the right elbow was excised for microscopic examination. Laboratory examinations at this time revealed nothing new except that secondary anemia had developed (red blood cells, 3,300,000; hemoglobin, 70 per cent). The patient was therefore given several transfusions during the next few weeks. The arthritis improved considerably, but the ocular condition did not. One month after the patient's admission the eye was so painful that a hypodermic injection was necessary. The globe was extremely tender to touch. It was becoming more congested in the affected region, and the sclera in the depressed area was apparently becoming thinner. On April 12, 1937, two months after admission, the eye showed the appearance depicted in figure 2. The lesions appeared to be about the same as when previously seen, except that now a nodule situated at the posterior margin of the cavity was observed.

The patient continually begged to have the eye removed. Enucleation was finally agreed on because of the fact that the scleral process was progressing, because of the severe ocular pain and because of the low visual acuity of the eye. It was feared that perforation of the globe might occur should the process progress much further. The left eye was enucleated on April 17, at which time the scleritis had existed certainly seven months and possibly fifteen months. The patient made an uneventful recovery from the operation and was discharged from the hospital one month later.

Histologic Examination of Left Eye.—The eye was fixed for forty-eight hours in a 4 per cent solution of formaldehyde and immersed for twenty-four hours in a solution of 2.5 per cent hydrochloric acid in 70 per cent alcohol. It was then opened (the retina was found to be in situ) and embedded in pyroxylin. Horizontal sections were made through the portion of the eye containing the lesions in the sclera and fundus. A part of the block containing the scleral lesions was later excised, embedded in paraffin and cut in serial sections. Most of the sections examined were stained with hematoxylin and eosin.

On microscopic examination the essential pathologic process was found to be confined to the anterior portion of the sclera and nowhere to extend farther back than 3 mm. behind the ora serrata. Sections passing through the lower two scleral nodules showed in the center of each nodule a large abscess about 2.25 mm. in length and 0.4 mm. in thickness (fig. 3). Each abscess consisted of a central mass of pus cells surrounded by a wall of epithelioid cells from four to six cells thick. The epithelioid cells were most often arranged radially with respect to the abscess, and among them there was an occasional giant cell of the Langhans' type. Most of the pus cells within the abscesses were necrotic, their nuclei showing chroma-

tolysis, pyknosis or fragmentation. Macrophages were not recognizable here or elsewhere in the tissues. In some sections remains of necrotic scleral tissue could be definitely recognized within the abscesses. The two abscesses were separated by a space of only about 0.5 mm.

Outside the walls of epithelioid cells many of the clefts in the sclera, especially those containing vessels, were distended with plasma cells and many fibroblasts. Some of the plasma cells showed colloid degeneration. Eosinophils were absent. The original sclera near the abscesses was being destroyed by the infiltrate and replaced by new fibrous tissue. There were few if any new vessels. Where the infiltrate was dense, the arteries showed marked obliterative endarteritis. The sclera was everywhere free from extravasated blood, blood pigment, edema and fibrin. Where the inflammatory process was marked, it also involved the underlying uvea, the stroma of which was here largely replaced by new fibrous tissue, chronic

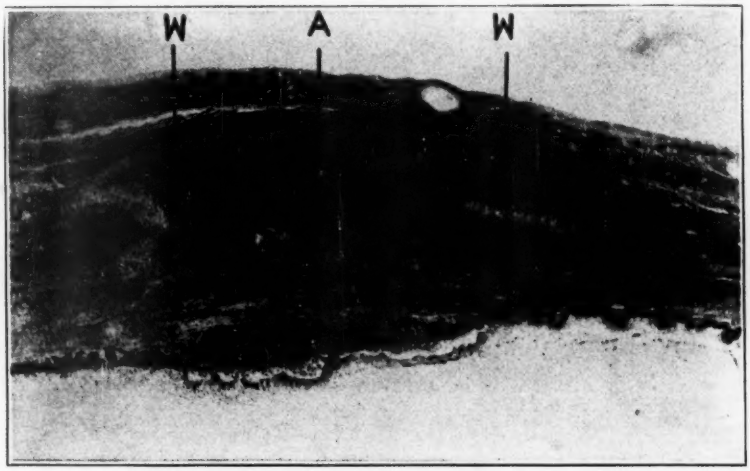

Fig. 3.—Photomicrograph showing a large abscess (*A*) in the sclera surrounded by a wall of epithelioid cells (*W*); × 27. The conjunctiva is absent here as the result of the enucleation.

inflammatory cells and fibroblasts, down to the unpigmented layer of ciliary epithelium. This epithelium was unbroken but was altered by edema and separated from the overlying tissue in the form of numerous small vesicles containing mononuclear cells and a few pus cells. The pigmented layer of ciliary epithelium had almost completely disintegrated, and great numbers of pigment cells were disseminated through the tissue. In some places, but never farther back than 1 mm. behind the ora serrata, the choroid had been disintegrated by the cellular infiltrate, which here had broken through the pigment epithelium and lifted up the retina. The latter, however, was only slightly altered. In spite of the marked chronic inflammatory reaction in the sclera and uvea, relatively few cells had exuded into the vitreous anywhere, and there was almost no tendency to the formation of cyclitic membranes.

In the affected region the whole thickness of the sclera proper was involved in the inflammatory process. The overlying episclera and conjunctiva showed slight edema, considerable congestion and infiltration with chronic inflammatory

cells, chiefly plasma cells. The entire ocular wall, even at the sites of the abscesses, was only about 1.5 mm. thick in the sections.

Sections passing through the upper two nodules showed essentially the conditions just described. One of the abscesses here was longer and narrower than any of the other large abscesses. It was noteworthy that almost no pus cells could be seen approaching any of the abscesses from outside their walls. In none of the sections was an abscess found which was being invaded by blood vessels and fibroblasts, that is to say, which was undergoing organization.

Sections passing through the cavity in the sclera showed a different picture (fig. 4). For a distance of about 3 mm. the conjunctiva and episclera were absent, and the sclera and underlying uvea had been largely replaced by fibrous tissue. This was coated externally with new epithelium and was only slightly infiltrated with inflammatory cells. The ocular wall was reduced to a thickness of only 0.1 mm. The tissue replacing the uvea was markedly pervaded with pigment cells derived from the original pigmented epithelium. The unpigmented layer of ciliary epithelium had not been broken through. Anteriorly and posteriorly the original

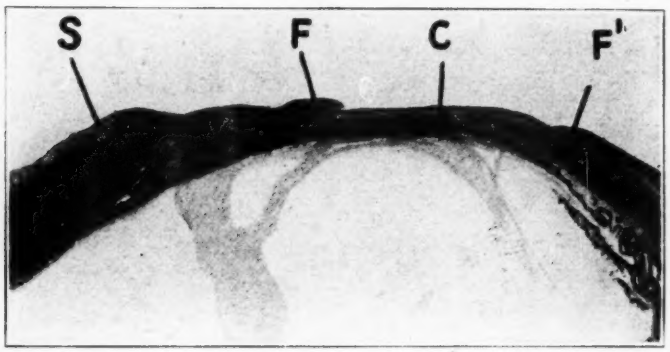

Fig. 4.—Photomicrograph showing a cavity (*C*) in the sclera coated with epithelium, flaps of conjunctiva (*F* and *F*[1]) extending over the margins of the cavity, and a disintegrating new sequestrum (*S*) situated posterior to the cavity; × 10. In some other sections the sclera is somewhat thinner at the site of the cavity.

conjunctival and episcleral tissue extended over the margin of the cavity in the form of a flap (fig. 4), the under surface of which was coated with new epithelium. This new epithelium extended for a long distance deeply into the new fibrous tissue, just as the surface epithelium may do in cases of indolent ulcers elsewhere, and at one place formed a small cyst.

Just posterior to the thinned area described there was a nodule which showed a still different stage in the inflammatory process. Here at about the middle of the sclera was a stretch of necrotic tissue about 3 mm. long and 0.25 mm. in its greatest thickness. It was easily recognizable as a sequestrum of sclera (fig. 4). It showed all stages in transformation from simple hyaline necrosis to almost complete granular disintegration. At the posterior end, where the sequestrum was hyaline and only slightly infiltrated, a wall of epithelioid cells was directly applied to it (fig. 5). Elsewhere the sequestrum was surrounded by, and was being invaded from, an irregular zone of pus cells. Around this the wall of epithelioid cells continued, but in some places it had been so disintegrated by the pus cells as to be

Fig. 5.—Photomicrograph showing the posterior end of the same sequestrum surrounded by a wall of epithelioid cells; × 85. At *S* the sequestrum is only slightly infiltrated and is not disintegrated, proving that the formation of the wall of epithelioid cells precedes the formation of an abscess.

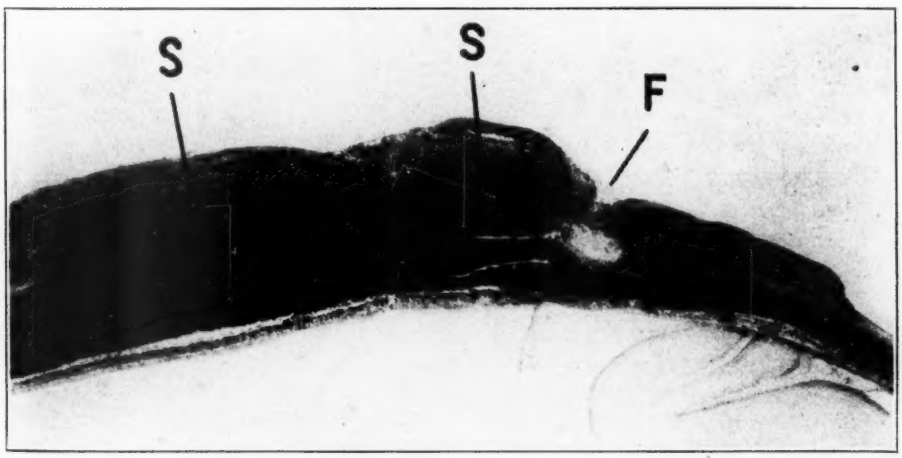

Fig. 6.—Photomicrograph showing a fistula (*F*) at the anterior end of same sequestrum (*S*); × 20.

recognizable only with difficulty. At its anterior end the sequestrum was greatly disintegrated, and from the cavity it occupied there extended to the surface through a break in the conjunctiva a small fistula (fig. 6). That this break was not an artefact was evidenced by the fact that the epithelium had proliferated downward and had begun to line the wall of the fistula. The entire fistula was included in a complete series of paraffin sections made through this region. In some sections the downgrowth of epithelium, extending from beneath the flap of conjunctiva, as previously described, reached the cavity of the sequestrum. A similar but much

Fig. 7.—Section near the horizontal meridian of the eye; × 55. *A* indicates a minute abscess near the filtration angle, surrounded by epithelioid cells, and *I*, an infiltrate in the region of Schlemm's canal. Note the absence of infiltration in the ciliary processes and the slight infiltration of the iris and ciliary body.

smaller sequestrum, not yet completely infiltrated, was found just beyond at the upper inner margin of the thinned area of the sclera.

In addition to the large abscesses, relatively minute foci, evidently of the same nature, were seen here and there within the affected region of the sclera. One such focus, situated near the canal of Schlemm, is shown in figure 7. This consisted of a small dense mass of necrotic pus cells, surrounded by a wall of epithelioid cells. It was evident that this focus had destroyed the scleral fibers here and not simply distended a scleral cleft. In some of these small foci remains of necrotic tissue could be seen. In several sections there was found near the abscesses a large intrascleral nerve loop surrounded, and to some extent invaded, by chronic inflam-

matory cells and fibroblasts. Several other large nerves, seen within the sclera and the anterior portion of the uvea in the affected region, were similarly involved.

In all sections the inflammatory process in the sclera ended posteriorly with notable abruptness; within a distance of 1.5 mm. it was marked and completely disappeared. Anteriorly it ended less sharply but did not extend into the cornea. The tissue of the limbus showed relatively slight infiltration, chiefly with plasma cells. The filtration angle was blocked by the root of the iris only opposite the excavated portion of the sclera. Where the angle was open in the affected region, the ligamentum pectinatum was in general only slightly infiltrated, chiefly with plasma cells. In many sections, however, the site of the canal of Schlemm was occupied by a dense cellular infiltrate, which often continued into the sclera along vessels communicating with the canal (fig. 7).

Elsewhere than in the immediate vicinity of the affected portion of the sclera, the ocular tissues were almost free from inflammatory reaction. Posteriorly to the lesions, the infiltration of the choroid soon became perivascular and completely disappeared at the equator. The retina showed insignificant perivascular infiltration, which also disappeared at the equator. Even where closest to the scleral abscesses,

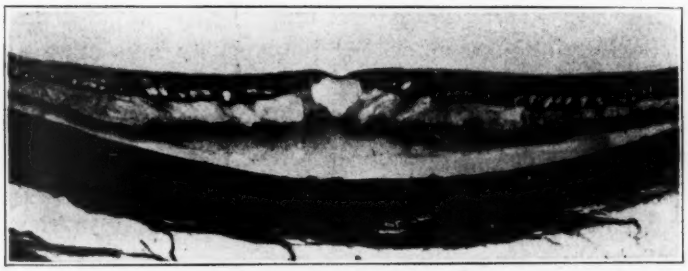

Fig. 8.—Photomicrograph showing the marked cystoid condition of the macula; × 20. The separation of the macula may be an artefact. There are two colloid excrescences almost directly behind the fovea.

the anterior part of the ciliary body and root of the iris showed only insignificant infiltration with plasma cells, and the ciliary processes were entirely free from infiltration (fig. 7). Elsewhere the iris was normal except for the presence within it of a few plasma cells. The ciliary body at a distance from the scleral foci showed an occasional small collection of lymphocytes. The retina showed unusually slight peripheral cystoid degeneration. There were many colloid excrescences scattered over the fundus. The optic disk and nerve stem were normal. The vitreous was free from cellular infiltration except in the immediate vicinity of the scleral lesions, and even here the cellular infiltration was slight, as already stated. The anterior chamber contained a delicate serous coagulum. Adherent to the posterior surface of the cornea were a few mononuclear cells, some of them in clumps of as many as six cells. These cells rarely contained pigment. The lens and cornea were normal. The central retinal vessels (examined in serial cross sections), the posterior ciliary vessels and the vessels of the choroid and retina showed slight if any endovasculitis.

In the fundus, at the site of the large pigmented area seen with the ophthalmoscope, the retina was atrophic and fused to the choroid. Neuroglia proliferated from the retina had here invaded the choroid. The histologic picture of this lesion was typical of an old completely healed focus of localized chorioretinitis of tuberculous origin.

In the macular region, involving an area about 6 mm. in diameter, the retina showed a cystoid condition, which at the fovea was extremely marked (fig. 8). Within the internal nuclear layer and Henley's layer there were numerous distended spaces. The largest were within Henley's layer, and these contained a delicate serous coagulum indicative of edema. There were also spaces within the ganglion cell layer, but these were small. The ganglion cells were apparently normal. There was no cellular infiltration or fibrin. The retina here was slightly separated from the choroid and considerably distorted, probably largely because of the fixation process.

Sections stained for tubercle bacilli, others stained by Verhoeff's modified Gram method, and still others by a variety of bacterial stains all failed to show any microorganisms within the abscesses or elsewhere in the eye.

COMMENT

The clinical appearances of the scleral lesions (figs. 1 and 2), their prolonged activity and their association with rheumatoid arthritis leave no doubt that the ocular condition in this case was that designated by van der Hoeve as scleromalacia perforans. The old healed focus of localized chorioretinitis, seen ophthalmoscopically and microscopically, and the old pulmonary lesions, revealed by roentgen examination, were no doubt tuberculous in origin and evidently unrelated to the scleritis; tuberculous scleritis has an entirely different histologic picture. Histologically, the large abscesses within the sclera were the most conspicuous active ocular lesions. They corresponded in position to the nodules observed clinically. Except in the immediate vicinity of the affected portions of the sclera, the eye was almost free from cellular infiltration. From the histologic appearances of the lesions, and especially from the appearance presented by the large sequestrum seen in one nodule, the steps in the evolution of an abscess were readily ascertained to be as follows: An area in the sclera first undergoes simple necrosis. This area soon becomes surrounded by a wall of epithelioid cells. Pus cells then slowly penetrate the wall and surround and infiltrate the necrotic area, which has become more or less disintegrated into eosinophilic granules. Ultimately the sequestrum thus formed becomes completely disintegrated and densely infiltrated with pus cells, which also become necrotic. That the process of abscess formation is extremely slow is evidenced by the fact that almost no pus cells are to be found outside the wall.

The size ultimately attained by an abscess is only slightly larger than the original area of necrosis. Large abscesses do not form from small ones except when the latter break into each other. There is slight if any distention of the sclera by an abscess. The elevation observed clinically is due chiefly to the inflammatory reaction around the abscess.

Only in our case, in Oast's case,[8] in van der Hoeve's first case and in Wojno's [9] case were scleral lesions other than the characteristic cavities seen. In these 4 cases nodules were seen in addition and recognized as being the precursors of cavities. It was noted that after a nodule was incised a cavity later existed at the site of the nodule. But in no case have clinical observations been made with sufficient frequency to show how, without operative intervention, transformation of nodules into cavities takes place. The histologic changes in our case, however, clearly indicate that usually the cavities result from abscesses perforating the conjunctiva and discharging their contents externally. They also show that at the sites of the ulcers thus produced the underlying sclera and uvea are largely replaced by a thin layer of newly formed fibrous tissue pervaded in its deepest portions by pigment cells and coated externally by new epithelium. In our case the original conjunctiva, evidently owing to its having been undermined, projected for a considerable distance over the margin of the cavity, and the under surface of the projection was coated with new epithelium.[10] No doubt in some cases the defect in the conjunctiva is so small that the edges later become approximated and the cavity completely covered with conjunctiva. Judging by the illustrations in Kiehle's case,[6] islands of conjunctiva may remain on a large cavity. At present there is no evidence that the abscesses produce cavities by undergoing organization or absorption.

In accord with the foregoing explanation as to the origin of the cavities in the sclera is the fact that in our case a small fistula was found extending from a sequestrum to the surface. Also in accord with it is the fact that there was no histologic evidence of absorption or organization of the abscesses. The fact that the abscesses showed no tendency to break into the interior of the eye was no doubt due to the great vascularity of the uvea.

It was evident in our case that while the major destruction of the sclera resulted from abscesses, some destruction also resulted from the chronic inflammatory reaction in the adjacent scleral tissue. The cellular infiltration occurred here primarily within the scleral clefts, especially in those containing blood vessels, and was obviously destroying the scleral tissue, which at the same time was being partially replaced by new fibrous tissue.

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8. Oast, S. P.: Scleromalacia Perforans: Report of a Case, Arch. Ophth. **17**:698 (April) 1937.

9. Wojno, Z.: Ein Fall von fortschreitender Erweichung der Lederhaut, Klin. oczna **13**:778, 1935; abstracted, Zentralbl. f. d. ges. Ophth. **35**:692, 1936.

10. Dr. Trygve Gundersen has recently had a patient with scleromalacia perforans who clinically presented exactly this condition. At the margin of a large scleral cavity a spatula was freely passed under the overhanging conjunctiva for a distance of about 1 mm.

In 2 of the previously recorded cases nodules were clinically regarded as containing abscesses. It was stated, however, in the report of van der Hoeve's case that when a nodule was incised it was found not to be an abscess. Probably this statement was based on the fact that fluid pus was not obtained. When one of the nodules was incised in our case, pus was obtained, as ascertained by microscopic examination.

In 2 cases,[11] scleral sequestrums were reported to have been seen within scleral cavities. One "sequestrum" was removed and on microscopic examination was found to consist of hyalinized scleral tissue free from cells.[11a] Judging by our microscopic observations, these "sequestrums," since they were entirely free from infiltration, could not have caused the cavities in which they were seen. Possibly they were simply degenerated scar tissue. As to whether or not a sequestrum can be spontaneously extruded before it has been completely infiltrated with pus cells, there is yet no evidence.

It seems possible that there may be cases of this condition in which some or all of the scleral lesions heal without producing the cavities supposed to be characteristic of the disease. In fact, one of us (F. H. V.) has recently seen what appeared to be undoubtedly such a case.[12] The patient, a woman aged 53, had well marked rheumatoid arthritis. In each eye the bulbar conjunctiva was everywhere intact, and the sclera showed many large bluish areas but no definite cavities. On the outer side of the right eye the bulbar conjunctiva was slightly congested and slightly elevated and showed beneath it a yellowish opaque substance occupying an area about 5 mm. in diameter. Presumably this was an area of scleral necrosis. Information was obtained that one year previously both eyes had been considerably congested for a long period and that there had been "episcleritis," descemetitis and mild iritis. This case strongly indicates that in the ocular disease under consideration, necrotic areas in the sclera, even when of large size, may in some cases be replaced by fibrous tissue without being infiltrated or extruded. Probably the case briefly described by Friedenwald [13] in 1921 was of this nature.

In the reports of none of the cases in the literature was ocular pain mentioned as a prominent feature, and in some of them it was stated to be absent. In our case it was severe and persistent. It was explained

11. (*a*) Urrets Zavalia, A.; Maldonado Allende, I., and Obregon Oliva, R.: Escleromalacia observada en el curso de una porfirinuria cronica, Arch. de oftal. de Buenos Aires **12**:115, 1937. (*b*) Soriano, F. J., and Riva, A.: Escleromalacia perforante, ibid. **12**:139, 1937.

12. The fact that one of us has seen within one year 3 patients affected with scleromalacia perforans suggests that this condition is far more frequent than the scarcity of its reported cases would seem to indicate.

13. Friedenwald, H.: Ocular Conditions Associated with Arthritis Deformans, Am. J. Ophth. **4**:431, 1921.

by the involvement of the anterior ciliary nerves in the affected tissues and particularly by the marked involvement of a large intrascleral nerve loop.

The marked cystoid condition of the macula in our case evidently resulted from edema due to the long-continued action of toxins arising from the inflammatory process in the sclera and diffusing through the vitreous. It was the great loss of central vision thereby produced and the continued pain that led us to acquiesce in the desire of the patient for removal of the eye. It seems probable that early curettage of the abscesses would prevent this edema of the macula and other possible intraocular complications.

At first glance, of the two designations for the disease under consideration, scleromalacia perforans, employed by van der Hoeve, and scleritis necroticans, employed by Rochat, the latter designation may seem to be preferable because it denotes the existence of scleritis. Since, however, it indicates that the scleritis causes the necrosis whereas the reverse is actually the case, it is really unsatisfactory. Unfortunately, scleromalacia perforans fails to indicate some of the important features of the disease. Thus it does not indicate that the primary process is necrosis, that it is focal, that there is inflammatory reaction with formation of nodules and that the sclera is excavated without necessarily being actually perforated. Necroscleritis nodosa excavans would describe the disease more completely and accurately. For cases in which there were no cavities, the term excavans could be omitted. Nevertheless, since van der Hoeve's designation has priority, has been generally accepted and is sufficiently distinctive, it seems best to retain it, at least until the exact cause of the disease has been ascertained.

A condition that bears some resemblance to scleromalacia perforans is brawny scleritis. Clinically, the two conditions are alike in their chronicity and in affecting the sclera predominantly. They differ in that brawny scleritis is more diffuse, does not cause definite nodules or cavities in the sclera and is not definitely associated with rheumatoid arthritis. Gilbert[14] in 1914, after microscopically examining an eye affected with brawny scleritis, contended, chiefly because the patient had gout, that this ocular condition was a manifestation of gout. Since, however, he was unable to find urate crystals in the eye, his case affords only suggestive evidence as to the cause of brawny scleritis. One of us has examined microscopically sections of five eyes supposed to have been affected with brawny scleritis.[15] The cases in which these eyes were removed were reported by Oatman, Parsons, Friedenwald, Schlodtmann and Verhoeff. In Oatman's case there was no necrosis; the

14. Gilbert, W.: Zur Aetiologie und pathologischen Anatomie der sulzigen Skleritis, Arch. f. Augenh. **76**:111, 1914.

15. Verhoeff, F. H.: Brawny Scleritis, Ophthalmoscope **11**:2, 1913.

infiltration, confined almost exclusively to the episcleral tissue and the uvea, was slight and not distinctive. In the other cases necrosis, although a conspicuous feature, undoubtedly was not the cause but a result of the inflammatory process. In Friedenwald's and Schlodtmann's cases the necrosis closely resembled the caseation of syphilis and tuberculosis. The possibility that brawny scleritis and scleromalacia perforans are due to the same cause, therefore, seems remote.

The problem as to the cause of scleromalacia perforans may now be considered. A possibility to be considered is that the scleral lesions are due to anemic necrosis, that is to say, to vascular obstruction. As to this possibility, we have at present no conclusive evidence. It is true that in our case obliterative endarteritis was marked in the vicinity of the abscesses, but it may well have been the result, not the cause, of the inflammatory process. On a priori grounds, it seems unlikely, although not impossible, that primary obliterative endarteritis, whether or not due to infection, would be confined to the particular sites and tissues in which the lesions of this disease occur.

In the present case, as in 2 previous cases, the bacteriologic investigations, including attempts at artificial cultivation and animal inoculations, yielded negative results. The histologic structure of the ocular lesions definitely excluded tuberculosis and syphilis. The abscesses in an advanced stage were consistent with a fungus infection, but, excluding from consideration vascular obstruction, in their formative stage did not suggest any process known to be infectious in origin.

A question of obvious importance is whether or not the nodules of scleromalacia perforans are similar to the subcutaneous nodules which not infrequently occur in cases of rheumatoid arthritis. In an attempt to answer this question one of us has examined sections of eight such nodules removed from different patients afflicted with rheumatoid arthritis.[16] One of these nodules was removed from the elbow of our patient. One nodule (not the one from our patient) showed in the connective tissue a large sharply defined area of necrosis, about 3 by 1.5 mm. in size. The necrotic area was intensely eosinophilic and almost completely disintegrated into granules of various sizes, from extremely minute to a size larger than a red blood corpuscle. At the periphery, the surrounding connective tissue could be seen undergoing rather abrupt transition into this necrotic material. In the contiguous tissues there were a few collections of coarsely vacuolated cells, apparently macrophages, the origin of which from connective tissue cells seemed obvious. There was no infiltration with lymphocytes or plasma cells. The impression given was that the process was more recent but more severe than in the other nodules. The latter showed

16. Dr. T. B. Mallory and Dr. W. Bauer, of the Massachusetts General Hospital, gave us the opportunity of examining these sections.

similar large areas of necrosis, but the necrotic tissue was far less granular. Most of the areas were completely walled off by epithelioid cells, just as were the scleral lesions in our case. In only two nodules was there considerable infiltration of the surrounding tissue with lymphocytes and plasma cells. In none of the nodules was there obliterative endarteritis or formation of new vessels around the necrotic areas. Within the necrotic areas irregular reticulated structures could often be seen. Each showed various transitions in staining, from intensely with hematoxylin to intensely with eosin. The impression given was that it was the cell syncytium of the original tissue, which in undergoing necrosis had been to a greater or less extent perfused with chromatin. Most of the necrotic areas showed insignificant infiltration with pus cells; some of them contained no pus cells. However, a large necrotic area in the nodule from our patient contained the largest number of pus cells, about one hundred in a high power field; but even this number was small compared to the number in the scleral lesions.

It is evident from the foregoing observations that the subcutaneous nodules of rheumatoid arthritis and the nodules of scleromalacia perforans in their initial stages are essentially alike. Why, as indicated by our case, the scleral nodules in their later stages tend to become densely infiltrated with pus cells and cause marked chronic inflammatory reaction while the subcutaneous nodules tend to remain relatively free from infiltration can only be conjectured. Possibly it is because of differences in the sensitivity of the affected tissues to the exciting agent. Trauma is undoubtedly an important contributory cause of the subcutaneous, but evidently not of the scleral, nodules.

Since the nodules of scleromalacia perforans and the subcutaneous nodules of rheumatoid arthritis are thus essentially alike, and since this type of scleritis is almost invariably associated with rheumatoid arthritis, it seems reasonable to suppose that the arthritis, the subcutaneous nodules and the scleritis are due to the same cause. It is evident that the initial scleral and the subcutaneous necrotic areas histologically resemble the necrotic areas of gouty lesions and do not resemble any lesions known to contain micro-organisms. In rheumatoid arthritis, areas of necrosis and in gouty arthritis, urate deposits occur in the cartilage of the affected joints. These facts suggest that the lesions of rheumatoid arthritis, including the subcutaneous and scleral nodules, are dependent on the deposition of some chemical substance in the connective tissues concerned. The relative avascularity of these tissues may be an important factor in the process. Chemical investigations of the lesions, by revealing the existence and nature of this chemical substance, may establish the fact that rheumatoid arthritis is due to some metabolic disturbance and even indicate the nature of the disturbance. Although the subcutaneous nodules of rheumatoid

arthritis are easily obtainable, apparently they have never been subjected to such investigation.

SUMMARY AND CONCLUSIONS

Fourteen cases of scleromalacia perforans described in the literature are reviewed, and a new typical case is reported, the first in which an eye affected with this disease has been obtained for microscopic examination. The patient was afflicted with rheumatoid arthritis.

From the histologic changes in this case it is evident that the primary ocular lesions each consists of a sharply defined area of necrotic scleral tissue. This area becomes surrounded by a wall of epithelioid cells and slowly infiltrated with pus cells. This process results in the formation of a sequestrum, which becomes completely disintegrated and densely infiltrated with necrotic pus cells.

The characteristic cavities in the sclera are generally due to abscesses so formed, perforating the sclera and discharging their contents externally. At the site of an abscess there remains a thin layer of newly formed fibrous tissue pervaded in its deepest portion by pigment cells. It is probable that there occur similar cases in which, without the formation of cavities, the necrotic tissue is replaced by fibrous tissue.

Through the long-continued action of toxins diffusing through the vitreous from the necrotic foci in the sclera, destructive edema of the macula may result. To avoid this and other possible intraocular complications, curettage of the scleral nodules is suggested.

Severe ocular pain of long duration may be a prominent symptom of the disease.

In the present case attempts to demonstrate micro-organisms in the abscesses by means of special stains, cultures and inoculation of animals failed.

The histologic structure of the lesions excludes tuberculosis and syphilis as possible causes of the condition.

Histologically, in their initial stages the scleral nodules are essentially similar to the subcutaneous nodules of rheumatoid arthritis. Their resemblance to the nodules of gout and their lack of resemblance to any lesions known to be due to direct infection suggest that the lesions of rheumatoid arthritis are dependent on the deposition of some chemical substance in the tissues concerned and hence that this disease is due to some metabolic disturbance.

ABSTRACT OF DISCUSSION

Dr. Frederick A. Kiehle, Portland, Ore.: A review of this case in connection with the others reported elsewhere calls attention to three points: (1) the comparatively short period of time the condition had existed, (2) the severe pain which the patient suffered and (3) the

minute abscesses which appear in the section. One might infer that the two last-named conditions—the abscesses and the pain—may be related as cause and effect.

The patient in the case which I reported at the 1936 meeting of the Academy of Ophthalmology and Otolaryngology, now bedridden for ten years, is still living; she is blind save for perception of light and is helpless due to arthritis but has a healthy heart and kidneys and good assimilation. The eyes show no further changes. No site of impending or of probable future perforation can be identified. As these cases, however, are often of many years' duration, the inference is that perforation is a late manifestation of the process.

In a recent personal communication Professor van der Hoeve confessed that he is unable to throw any new light on the subject, while in a communication from Sir Stewart Duke-Elder a recent case which he has observed is described. I quote from his letter:

"The patient with scleromalacia whom I saw in March was a woman of 56 who had been suffering from polyarthritic rheumatism for about thirty years and is now a complete cripple with most of her joints ankylosed. The right eye showed two large areas over which the sclera had completely disappeared, exposing the uvea, and in the left eye all around the ciliary region a similar condition existed, there being only one or two bands of sclera remaining. In the left eye there was a circular ulcer all around the cornea of an atrophic indolent type. Nothing in the picture suggested inflammation. I am proposing to do a mucous membrane graft from the lip to cover the exposed areas sometime in the near future."

It is only by calling attention to the existence of this disease, now attaining recognition as an entity, that these sporadic cases will be garnered into some sort of a whole from which definite conclusions can be drawn.

The evident association between scleromalacia and polyarthritis is a marked feature of the condition and one which continues to await explanation.

DR. SAMUEL P. OAST, New York: In the April 1937 issue of the ARCHIVES (page 698) I reported a case in which the ocular picture was strikingly similar to that in the case so painstakingly investigated and so ably presented by Dr. Verhoeff and Dr. King but in which there was no history of any condition resembling arthritic involvement; my patient was a robust appearing man of 76. The ocular condition was unilateral, and the patient did not have arthritis.

I have two slides made from drawings which I believe give an accurate idea of the condition as it was observed.

The first slide shows one of the characteristic nodules at about the height of its active stage before discharging its necrotic center. This drawing was made late in the course of the disease, so that the edges of the older healed foci may be seen to either side.

The second slide shows the sites of the nodules after healing had taken place, the blue uvea being evident through the residual scleral and cicatricial tissue. The eye remains in this state today, with the exception that all evidence of redness has disappeared.

In the 14 cases dealt with here there are two outstanding features, the most striking and common of which is the scleral thinning. The other is the localized inflammatory nodule. This was observed in but 4 of the 14 patients. Now whether or not some of the other 10 patients came under observation after the nodules had been present but had disappeared is a thought which has to be considered, but surely Dr. Kiehle's patient, who was under more or less continuous observation by him and other prominent ophthalmologists for over a period of years, did not have any localized inflammatory lesions. The fact stands out, therefore, that in some of these 14 patients inflammation was either absent or was not a prominent feature.

The different behavior of the condition in these two groups suggests to me that more than one etiologic factor may be responsible and that one may be classifying these possibly different conditions as one disease entity solely because of their similar end effects, as evidenced by absorption of the sclera.

I am therefore in accord with Dr. Verhoeff and Dr. King in objecting to the term "scleromalacia," particularly as applied to the type of lesion depicted in their report, for the principal reason that the term "scleromalacia" does not carry with it any thought of inflammation in the customary acceptance of that term.

I feel that I should not let this occasion pass without mentioning a circumstance in the treatment of my patient, the evaluation of which I shall leave to your judgment.

The usual symptomatic local measures had been followed for four months or more, and nothing that was done seemingly had the slightest effect on the unrelenting progress of the disease. I had all but despaired of saving the eye, when, skeptically, I began treatment with ultraviolet rays. Almost immediately the pain became less (pain had been an annoying feature from the onset), the old lesions began to disappear and no new nodules formed. I have since kept the patient under observation for nearly two years, and the eye has remained quiet clinically ever since.

I should like to ask Dr. Verhoeff whether, in view of the roentgen evidence of a healed, possible tuberculous lesion of the chest, the microscopic evidence of a tuberculous choroidal lesion and the finding in the scleral nodules of a broken-down center with a surrounding zone of epithelioid cells and some Langhans' type of giant cells, he has dismissed tuberculosis categorically from further consideration as a causative agent in this particular type of lesion.

DR. ARTHUR BEDELL, Albany, N. Y.: The presentation of a case of scleromalacia is of considerable interest because of the rarity of the disease and the thoroughness with which the authors have reported the case.

About two years ago a 70 year old woman was sent to me by Dr. Marshall of Rutland, Vt., with a history that her right eye had been red for six months, with what appeared to be scleritis and several marginal ulcers. The latter responded to treatment with trichloroacetic acid. Three weeks ago the scleral area became more purple, the overlying conjunctiva disappeared, and an ulcer developed.

The patient was otherwise in good health, never having suffered from any serious illness or local ocular disease.

When I examined her, vision in the right eye was 10/200. There was moderate conjunctival congestion with a few flat discrete follicles. The pupil was 3 mm. in diameter and regular, with a sluggish reaction to light. There were several small, deep corneal ulcers, 0.5 mm. in diameter, near the upper limbus, and the upper one fifth of the cornea was rough, gray and infiltrated. Four millimeters from the superior limbus was a large, oval, bluish black crescent, 10 by 5 mm., with depressed, rounded borders. The floor of the ulcer, a dark, almost black, protruding soft mass, like a choroidal prolapse, was divided into two parts by a gray band 2 mm. wide. The nasal half was a little the larger.

The clinical course was protracted. At times the eye became almost white, the cornea became smooth, and the scleral ulcer lost some of its dark, bulging protrusions, but never flattened. The conjunctival overgrowth and the scar tissues reduced the surface loss, but the corneal infiltration became much greater and the vision was reduced to perception of light.

The eye was such a constant source of irritation that it was enucleated eight months later.

The sclera was thin, beneath an overhanging conjunctival shelf. The infiltration of the cornea was so great that the only portion which remained clear was a small central opening.

The left eye was normal.

General examination gave negative results. Smears and cultures gave no clue to the cause.

The patient had no evidence of rheumatoid arthritis.

(Several lantern slides of direct color photographs of the fundus were used to illustrate the discussion.)

DR. TRYGVE GUNDERSEN, Boston: I have some colored photographs of the eyes of a patient who has been under my care for the past two and a half years. The patient was first seen in November 1935 at a convalescent home, where she was being treated for severe rheumatoid arthritis. She complained of sore eyes. A diagnosis of chronic conjunctivitis was made.

She was seen again about a month later, when acute glaucoma was present in each eye, and the tension was 50 mm. (Schiötz). The pupils were dilated. The anterior chambers were deep. There were no aqueous cells or keratitis punctata. The patient was immediately transferred to the Massachusetts Eye and Ear Infirmary. Paracentesis was performed on the right eye and iridectomy on the left eye. A complete separation of the choroid occurred in the left eye, which lasted for the ensuing three months. For three or four months the right eye continued to show a tension of between 40 and 50 mm. (Schiötz), which gradually subsided.

Scleritis developed in both eyes about four months after the onset of the glaucoma. Although the patient was seen at frequent intervals in the convalescent home, at no time could it be said that there was a definite discharge from any of the scleritic nodules. The undermining of the conjunctiva was such that a Bowman probe could be

passed under the overhanging edge of conjunctiva for a distance of 2 mm. At no time was there any ectasia of the ciliary body or uvea. The intraocular tension is now 10 mm. (Schiötz) in each eye.

Dr. Lloyd Mills, Los Angeles: I wish to take exception to a statement made in most of the texts, that no local treatment is of avail in the various forms of scleritis. About twenty-four years ago, when working in Fuchs' clinic, I was struck by the constrictive effect of a 20 per cent solution of zinc sulfate when applied to lesions about the limbus. This application often led to the healing of phlyctenules. It was but a step to apply this, with considerable success, in cases of episcleritis, scleritis and brawny tenonitis, always in connection, of course, with the elimination of infective or other allergens wherever possible.

The element of chronicity appears to be the element of importance in these cases. The examination of sections from such eyes show them packed with lymphocytes, the tissue spaces being filled with edema, and it is only logical that continued increased tissue pressure of this sort may provide the basis for necrosis.

It has seemed to me, therefore, that if the time of healing could be hurried, there would be far less chance for these serious sequels. For over twenty years I have been using a 20 per cent solution of zinc sulfate in all cases of scleritis, regardless of the clinical background or severity of the condition. It definitely has hastened the arrest of the lesions and has lessened or prevented scarring.

The application is made as follows: A 4 per cent solution of cocaine hydrochloride and epinephrine in a dilution of 1:1,000 are instilled four times. In the first treatment there is little or no blanching. Then a swab, wet, but not dripping, is applied to the margin of the lesion and kept steadily there for twenty seconds. The entire circumference of the lesion and its center are similarly covered, with special attention to the blood vessels. The eye is finally washed with boric acid, and cold compresses are applied until the patient is comfortable. Treatment is repeated daily. Usually on the third or fourth day of treatment the lesions begin to blanch, become defined and gradually flatten, with scars, if of long duration, and without scars, if acute.

Dr. Frederick H. Verhoeff, Boston: Dr. Kiehle brought up the question of the short duration of the condition in the case reported. Since in previous cases there is no evidence as to how long a time is required for a nodule to come and go, I do not think that his point is well taken.

As to the question of pain, according to the reports it is seldom mentioned, and I think that was an unusual feature in this case. It was explained well, however, by the fact that we found ciliary nerves much infiltrated in the region of the lesions.

Dr. Oast suggests that in some cases this scleral process may occur without its having been preceded by a nodule. I called attention to the possibility that the condition in all cases may not be like that observed by us; in fact, I recently saw a patient in whom the sclera showed these bluish areas of discoloration, with the conjunctiva intact over them and no depression whatever. I think that such instances might often be overlooked.

Dr. Oast suggests that the condition is not an entity; it may be due to various causes. This seems to me unlikely, because the ocular condition is so much alike in all the cases and in the great majority is associated with rheumatoid arthritis.

Dr. Oast also brings up the question of tuberculosis, because I mentioned the occurrence of giant cells, epithelioid cells and necrosis. But these are found in a good many conditions. It is not their occurrence but their arrangement that is of chief diagnostic significance. No general pathologist, I am sure, would think for a moment that the lesions in this case were tuberculous. Moreover, if they were tuberculous, which I cannot conceive of, the material in the abscesses would have been just the sort of material that would infect a guinea pig. We injected the material into a guinea pig and did not produce tuberculosis. We also injected the material beneath the conjunctiva of a rabbit with negative results.

The condition in Dr. Bedell's case may or may not be scleromalacia perforans. I think that it perfectly well could be. The corneal involvement is unusual, but it seems to me that it should be expected, judging from the pathologic changes, that the cornea should be involved in some cases in the way he describes.

Dr. Gundersen's case is the only one in which glaucoma was noted, and his case was interesting to me because it confirmed my observations regarding the formation of the excavations.

USE OF SORBITOL IN GLAUCOMA

JOHN BELLOWS, M.D.

IRVING PUNTENNEY, M.D.

AND

JACK COWEN, M.D.

CHICAGO

The lowering of the intraocular pressure by osmotic changes produced by the intravenous injection of hypertonic solutions is well known. Cantonnet [1] demonstrated that the ocular volume diminished after the intravenous injection of hypertonic salt solution. Hertel [2] showed in animals that the decreased tension produced by injections of hypertonic saline solution was accompanied by a decreased water content of the eyes. The clinical usefulness of the hydrophilic character of hypertonic solutions has found application in various fields of therapy, including that of ophthalmology. However, many ophthalmologists have not taken full advantage of osmotic therapy.

The possession of an additional means of lowering intraocular tension when methods ordinarily used either are contraindicated or are inadequate is a valuable asset. This is particularly apparent when intraocular tension cannot be controlled immediately after an operative procedure on the eye; in this case inflammation and tenderness are so great that neither additional operative intervention nor local ocular medication is tolerated. The further desirability of the use of a hypertonic solution intravenously can be demonstrated by the dangers and shortcomings inherent in the accepted surgical procedures for glaucoma.

Paracentesis, the simplest method for lowering intraocular tension, is fraught with the dangers of prolapse of the iris and injury to the lens and is followed, as Kronfeld [3] has shown, by a secondary rise in tension to a point higher than the initial tension.

Posterior sclerotomy, in addition to the dangers listed under paracentesis, adds the hazards of injury to the retina and choroid with possible detachment of the retina, injury to the uvea with ensuing sympathetic ophthalmia, intraocular hemorrhage following the chance

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From the Department of Ophthalmology, Northwestern University, and the Illinois Eye and Ear Infirmary.

1. Cantonnet, A.: Arch. d'opht. **24**:193, 1904.
2. Hertel, E.: Arch. f. Ophth. **88**:197, 1914.
3. Kronfeld, P. C.: Tr. Am. Ophth. Soc. **22**:115, 1928.

perforation of a retinal or large choroidal vessel and the introduction of infection.

The injection of procaine hydrochloride and epinephrine hydrochloride deep into the orbit adds to the danger of retrobulbar hemorrhage and proptosis the possibility of injecting material intravenously with serious consequences. The tension is often not affected by this means and may rise even higher.

Medical control of ocular hypertension also presents difficulties, for which intravenous hypertonic therapy is a solution. Miotics may be undesirable because of the possible formation of synechiae in those cases in which an exudative process is present. The surface application of epinephrine hydrochloride has its limitations: The patient must be watched for systemic reactions, since hypertensive persons may show an alarming rise in arterial tension; and, as previously mentioned, a paradoxic rise in ocular tension may occur. Purgatives and agents to produce sweating may be conceivably used, but both have undesirable side-effects in older and debilitated persons. In addition, Duke-Elder [4] has shown in a comparison of the effects of hypertonic solution given orally and intravenously that the osmotic effect produced by administration by the intravenous route is far greater than that produced by administration by the oral route. Osmotic therapy may be of great value as an emergency measure at a time when the services of an ophthalmologist are not available.

Hypertonic solutions in the control of ocular hypertension finally have their greatest usefulness in those cases in which the tension remains high in spite of the administration of the strongest miotics. Such therapy is a boon to the ophthalmologist who is faced with the necessity of operating on a stony-hard glaucomatous eye with all the attendant hazards, especially the hazard of intraocular hemorrhage.

Osmotic therapy, however, also has its disadvantages: There is danger of overburdening the heart with the increased blood volume, and injury to the median nerve may result. Specific objections can be raised as to the substances used for osmotic therapy. Sodium chloride is limited in its use because of its great diffusibility. The salt enters into the tissue spaces of the body, causing a general water-logged condition. This is most feared in those patients with cardiac and renal damage in whom chlorides are already retained and an edematous state is already present. In a like manner the salt enters the anterior chamber of the eye and causes imbibition of water with a secondary rise in tension to a point higher than the original tension. Furthermore, the injection of sodium chloride is in itself painful, and it produces a severe slough if extravasated into the tissue. However, it has the

4. Duke-Elder, W. S.: Brit. J. Ophth. **10**:1, 1926.

advantage of producing the greatest osmotic effect of any of the substances compared for the quantity used. It owes its great osmotic action to the small molecular size and marked ionizing power.

Dextrose, like sodium chloride, because it is readily diffused into the tissues produces a secondary rise in tension after an initial drop. This diffusibility of dextrose and chloride into the anterior chamber has been demonstrated by Duke-Elder and others, who found in the aqueous humor increased quantities approximating those in the blood stream after intravenous injections of these substances. A further objection to dextrose is that its use is limited to nondiabetic patients.

Sucrose, a nondiffusible disaccharide, was introduced to supplant the difficulties found with the use of sodium chloride and dextrose. Because of its large molecular size, greater quantities of this substance are necessary than of a monosaccharide. Experiments on animals show that this sugar does not enter the ocular tissues.

TABLE 1.—*Data on Sugar Content of Aqueous Humor*

	Before Injections, Aqueous Humor, Mg. per 100 Cc. of Sugar	After Injection of 30 Cc. of 50% Sucrose Intravenously, Aqueous Humor, Mg. per 100 Cc. of Sugar
Dog 1	79	80
Dog 2	81	80

EXPERIMENTAL WORK ON SUGAR CONTENT OF AQUEOUS

The aqueous humor of the right eyes of two dogs weighing from 35 to 40 pounds (15.9 to 18.1 Kg.) was aspirated, and the amount of sugar was determined. Then 30 cc. of a 50 per cent solution of sucrose was injected intravenously. After one hour the aqueous humor of the left eye was aspirated. After hydrolysis the total amount of sugar was determined. Table 1 shows that there was no increase in the reducing sugar. This points to the nondiffusible character of sucrose.

For these reasons sucrose seemed an ideal choice for osmotic therapy, since it accomplished its purpose without a secondary undesirable effect. Masserman [5] had already found that sucrose did not enter the cerebrospinal fluid and therefore did not occasion the secondary rise in intracranial pressure found with dextrose and saline solution. Dyar and Matthew [6] obtained favorable results with sucrose in a series of glaucomatous patients. Sucrose had been used by us prior to this report with good results but had been abandoned because of the findings

5. Masserman, J. H.: Bull. Johns Hopkins Hosp. **57**:12, 1935.

6. Dyar, E. W., and Matthew, W. B.: Use of Sucrose Preparatory to Surgical Treatment of Glaucoma: Preliminary Report, Arch. Ophth. **18**:57 (July) 1937.

of H. A. Lindberg,[7] of Northwestern University, who presented evidence of severe renal damage in dogs after its use. This damage consisted of swelling of the collecting tubule to apparent closure and of inflammation of the glomeruli, with later hyalinization in some, indicating permanent damage. After the intravenous injection of sucrose in human beings Lindberg found occasional blood cells and casts in the urine.

Because of the objectional features of the aforementioned agents which have been used up to now for osmotic therapy, it would be of great value if a substance could be found which would be relatively nondiffusible, nontoxic, effective in moderate quantities and usable in persons with diabetes. Of all the agents which are readily available, sorbitol [8] most nearly satisfies these requirements.

Sorbitol is chemically a complex alcohol of approximately the same molecular size as dextrose. West and Carr,[9] who have made extensive studies of its toxicity, find it less toxic than sucrose, while Lindberg has found that the kidneys are not altered in the slightest degree by it. Although data regarding the metabolism of sorbitol are yet incomplete, the evidence points to its inertness, and therefore it may be safely used in patients with diabetes. In Germany it is used as a sweetening agent by diabetic persons. For these reasons it occurred to us that this compound would be better than any substance heretofore used for osmotherapy in glaucoma.

EXPERIMENTAL WORK WITH SORBITOL

In order to determine the amount of diffusibility into the anterior chamber of the eye, experiments were carried out in which sorbitol was injected intravenously into dogs in amounts slightly greater relatively to the amount used in human beings. It was found that only small amounts (from 5 to 10 mg. per hundred cubic centimeters) entered the anterior chamber of the eye. (Further work along this line is now being carried out and will be reported later.) Therefore it appears that sorbitol is far less diffusible than dextrose and sodium chloride, and from this it follows that it would not produce as marked a secondary rise in tension as the latter substances.

The clinical investigation was undertaken by us to determine its value in patients with increased intraocular tension (including those with primary and with secondary glaucoma). One hundred cubic centimeters of the 50 per cent solution was administered intravenously. All liquid foods were restricted on the day of this study. Small sips of water were permitted to ease the thirst. Part of the series received no miotics from twelve hours prior to the injection until the end of the period of observation. The tension of the patients was determined at intervals of two hours day and night by the same observer and with the same

7. Lindberg, H. A.: Personal communications to the authors.

8. Abbott Laboratories supplied the material.

9. West, E. S., and Carr, C. J., cited by Potter, R. D.: Science (supp.) **87**: 8 (April 29) 1938.

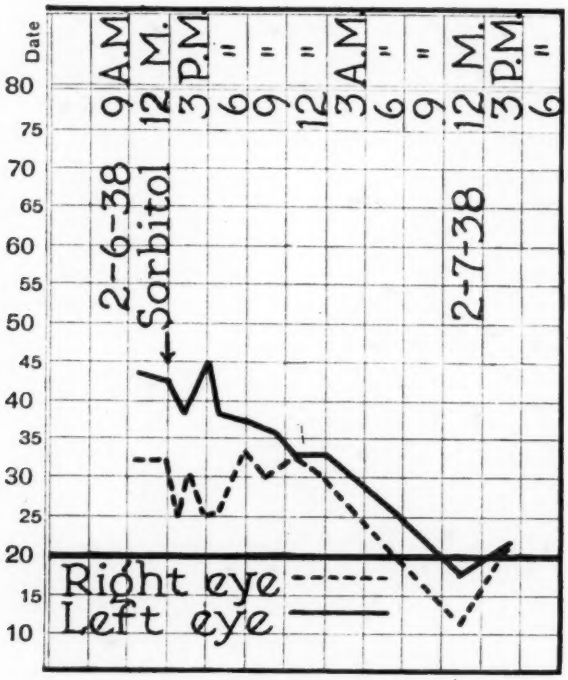

Chart 1.—Tonometric chart of P. S.

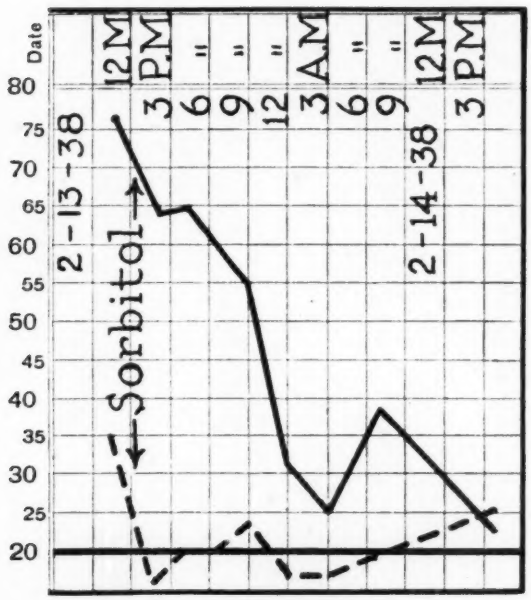

Chart 2.—Tonometric chart of F. L.

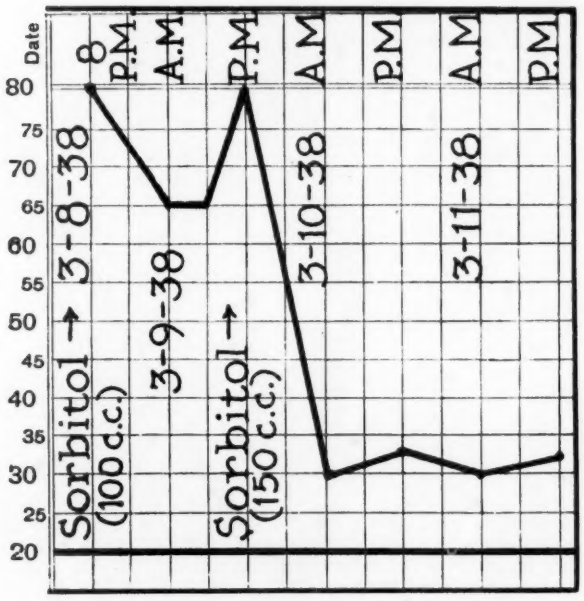

Chart 3.—Tonometric chart of M. H.

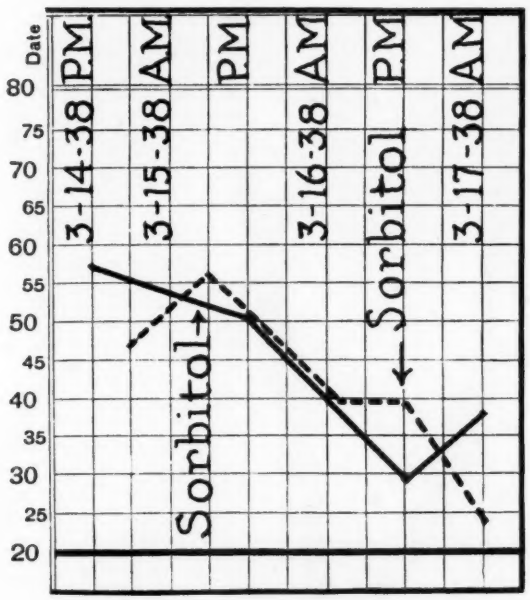

Chart 4.—Tonometric chart of F. M.

TABLE 2.—*Effect of the Intravenous Injection of Sorbitol on Intraocular Tension*

Patient	Diagnosis	Intraocular Tension, Mg. of Hg (Schiötz)															
		Before Administration of Sorbitol	After Administration of Sorbitol, Hours														
			2	4	6	8	10	12	14	16	18	20	22	24	26	36	48
J. K.	Congestive glaucoma	40	38	38	33	34	34	34	..	..	..	..	20	..	35		
P. S.	Right eye, chronic glaucoma	44	40	38	38	36	34	34	..	..	24	..	17	..	22	(chart 1)	
	Left eye, hemorrhagic glaucoma	32	30	26	32	30	34	32	..	..	20	..	12	..	22		
F. L.	Glaucoma simplex																
	Right eye	37	17	20	20	23	..	17	..	17	20	..	..	..	23	(chart 2)	
	Left eye	77	65	65	58	55	..	32	..	25	38	..	..	..	25		
N. P.	Glaucoma simplex	44	38	38	37	23	..	25	..	25	..	38	..	..	41		
T. B.	Chronic glaucoma	44	32	35	37	38	..	..	25	..	35	..	..	..	..		
O. N.	Bilateral glaucoma simplex																
	Right eye	44	44	44	32	44	35	..	35	..	44	..	32	..	..		
	Left eye	50	52	48	37	48	48	..	48	..	48	..	30	..	..		
C. G.	Left eye, glaucoma capsulare																
	Right eye	17	20	20	16	16	18	16	..	..	12	..	12	20	..		
	Left eye	32	28	28	33	32	30	34	..	..	24	..	17	30	..		
M. H.	Intumescent cataract; secondary glaucoma	80	..	..	..	..	..	65	..	..	65	..	..	83	..	(chart 3)	
		83	(Sorbitol repeated)					30	..	..	..	..	..	..	..	32.0	30
G. J.	Glaucoma simplex (diabetic)	51.5	..	..	..	..	..	38	..	..	..	..	..	38	..	32.5	
F. M.	Glaucoma simplex (chart 4)																
	Right eye	55.0	59.5	..	..	51.5	..	..	..	..	..	41.5	(Sorbitol repeated)			23.5	
	Left eye	51.5	51.5	..	..	51.5	..	..	..	..	..	41.5				38.0	
H. S.	Glaucoma after cataract	38	..	27	25	..	..	23	41	..	..	..	38	(Sorbitol repeated)		....	17
M. P.	Glaucoma simplex (normal)																
	Right eye	64	..	..	..	..	..	44	..	55	(Sorbitol repeated)			44	..	....	23
	Left eye	20	..	..	..	..	..	12	..	12				13			

Schiötz tonometer, with the exception of a few on whom this procedure could not be carried out.

RESULTS

In all cases in which sorbitol was administered, lowering of the intraocular tension was observed (table 2 and charts 1-4). The greatest dramatic drop was obtained where it was most desirable, namely, in patients with high intraocular tension who do not respond to the administration of miotics alone. Although a gradual decrease in tension was noted as early as two hours after the injection, in most cases the maximal effect was reached from twelve to twenty-four hours after injection. Then the tension gradually rose and reached a level approaching the original value, but in no instance did the tension rise appreciably above that point. In several instances in which operative procedures were not undertaken the intraocular tension observed for a number of days remained at a fairly normal level. In some persons repeated injections of sorbitol achieved a pronounced effect when a single injection had produced little change.

SUMMARY AND CONCLUSIONS

Sorbitol was investigated with a view to replacement of less desirable agents in osmotic therapy. Laboratory experiments on dogs show sorbitol to be slightly diffusible into the anterior chamber as compared to dextrose and sodium chloride. It was found to be an effective agent in reducing abnormally high intraocular tension when injected intravenously into human beings. Because of its slight diffusibility, a secondary rise in tension above that of the initial value was not observed.

In conclusion, we recapitulate that 100 cc. of a 50 per cent solution of sorbitol administered intravenously and repeated in twenty-four hours, if necessary, will nearly always reduce intraocular tension when it is greatly increased and is not controlled by the use of miotics alone. It thus relieves the attending pain and prepares the eye for operation.

Clinical Notes

A NEW REFRACTOR SUSPENSION

S. MAISLER, M.D., SAN FRANCISCO

The instrument here illustrated affords a new type of refractor suspension. It embodies new streamline designing, unity, ease of operation, facility of arm and chin rest for the patient and a writing table for the examiner.

A new refractor suspension.

The black crackle-finished tubular suspension can be utilized for any type refractor; it is mounted on an electric lift table, which requires only the slightest pressure on the button switch for quick, smooth and easy vertical adjustments. Gross adjustments for the refractor in the lateral and anteroposterior directions are adequately controlled by the movement of the entire table, which is mounted on ball-bearing casters. The finer adjustments in the anteroposterior position of the refractor are made with an adjusting screw of the refractor itself.

The vitrolite-covered table provides spacious writing facilities for the examiner and affords a resting place for the patient's elbows and forearms. The chin rest also enhances the comfort of the patient. A retinoscope and ophthalmoscope with cord reels and the necessary rheostat are easily added accessories.

The table will also be available in offset pedestal design, to be used in conjunction with an ophthalmic chair.

This new suspension unit was developed with the assistance of Mr. A. Parsons, of Trainer and Parsons, San Francisco.

350 Post Street.

A NEW CAPSULE-GRASPING FORCEPS

M. M. CULLOM, M.D., NASHVILLE, TENN.

A new capsule-grasping forceps for use in the intracapsular extraction of cataracts is here described. It is a double-headed forceps, designed to grasp the capsule at two points. The advantages are obvious.

Fig. 1.—A new capsule-grasping forceps.

Fig. 2.—Head of the forceps, showing the prongs closed.

Surgeons who use a capsule-grasping forceps say that in about 50 per cent of the cases the forceps tears out, necessitating extracapsular extraction. When the capsule is grasped at two separate points the chances of this occurrence are greatly lessened and there is much better leverage, so that it is easier to dislocate the lens and bring it out intact in the grasp of the forceps.

The forceps should, of course, be closed when it is inserted into the eye, and then be allowed to spread. The capsule can be grasped and the necessary rocking movements made to dislocate the lens. There is a natural spread of 4 mm. between each pair of blades.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

ETIOLOGY OF RETINAL SEPARATION CONSIDERED FROM THE STANDPOINT OF SURGICAL CORRECTION

EDMUND B. SPAETH, M.D.

PHILADELPHIA

In this review regarding the surgical correction of retinal separation certain factors are considered which must be of etiologic importance, judging from the successes and failures resulting from present day operations. They are presented without any attempt at correlation save their apparent pertinence. It is even impossible to estimate the relative importance of some of these various factors, one as compared with the other. The only certainty is that all are relevant.

HISTORY

The modern treatment of separation of the retina is perhaps the outstanding single advance in ophthalmology within the present generation. Even this period, relatively short as it is, is definitely narrowed, in that the earliest article which initiated the recent research (and the tremendous strides which have been achieved) is that by Verhoeff [1] in 1917. From this time until Larsson's [2] work in 1928 and in 1930, Vogt's [3] in 1929, Sourdille's [4] in 1929 and Gonin's [5] in 1930 the literature was rather sterile of satisfactory operative procedures. To be true, many discussions of the etiology, theories as to formation and considerations of the pathologic picture were presented in this interval, but for some reason the general ophthalmologic world seemed dormant to the marvels which were occurring, at least unfolding, before them. Even early articles connected with the surgical treatment passed unnoticed until attention was called to them by the tremendous amount of literature which suddenly appeared after 1930. Some of these early articles

From the Peter Clinic, the Graduate School of Medicine, the University of Pennsylvania.

1. Verhoeff, F. H.: Ophth. Rec. **26**:10, 1917.
2. Larsson, S.: Acta ophth. **3**:319, 1926; **6**:344, 1928; **8**:172, 1930.
3. Vogt, A.: Klin. Monatsbl. f. Augenh. **82**:619, 1929.
4. Sourdille, G.: Prat. méd. franç. **8**:122, 1929.
5. Gonin, J.: Ann. d'ocul. **167**:361, 1930.

included that already mentioned by Verhoeff,[1] Gonin's [6] work of 1919 and of 1921, Lenz's [7] of 1922, Sourdille's [8] of 1923 and Jocqs's [9] of 1920.

It is interesting that the modern basis for the surgical treatment of retinal detachment had been considered long before; Anderson [10] in his excellent bibliography on the subject called attention to the "formation of adhesions" with such treatment by citing an article by James Ware [11] on electrolysis and articles by Clavelier and Maravel [12] and Terson; [13] the provision for permanent drainage was emphasized as early as 1872 by de Wecker.[14]

Vogt,[15] with the help of his assistant Latte, was able to present the following definite dates:

Who was the first to see a retinal tear? Coccius,[16] in 1893. . . . The next was A. von Graefe,[17] who, however, regarded the tear as a part of the healing process, until Hansen [18] in 1871 and Schweigger [19] in 1873 advanced the opposite view.

Who recognized for the first time that the tear was the cause for the detachment? De Wecker [20] of Paris, in 1870. In his book he described three types of retinal detachment: "Décollement par distension, par attraction et par soulèvement" (detachment by distention, by attraction and by swelling). In the first two, according to him (1870) the retinal tear is the direct cause for the detachment. In 1882, Leber [21] popularized this mode of development. According to him (1882), every acute detachment which can be recognized with the ophthalmoscope is caused by a retinal tear

6. Gonin, J.: Cor.-Bl. f. schweiz. Aerzte **49**:1675, 1919; Ann. d'ocul. **158**: 175, 1921.

7. Lenz: Augenärtzliche Operationslehre, in von Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1922, p. 1289.

8. Sourdille, G.: Arch. d'opht. **40**:419, 1923.

9. Jocqs, R.: Clin. opht. **24**:357, 1920.

10. Anderson, J. R.: Detachment of the Retina: A Contribution to the Study of Its Causation and Treatment, New York, The Macmillan Company, 1931.

11. Ware, J.: Chirurg **2**:238, 1805.

12. Clavelier and Maravel: Ann. d'ocul. **115**:37, 1895.

13. Terson, A.: Ann. d'ocul. **114**:22, 1895.

14. de Wecker, L.: Ann. d'ocul. **67**:137, 1872.

15. Vogt, A.: Operative Treatment of Detachment of the Retina: Abstract of the Official Review Presented Before the International Ophthalmological Congress, Madrid, Spain, April 18, 1933, Arch. Ophth. **10**:293 (Sept.) 1933.

16. Coccius, A.: Ueber die Anwendung des Augenspiegels, Leipzig, I. Müller, 1853, p. 131.

17. von Graefe, A.: (*a*) Arch. f. Ophth. (pt. 1) **1**:358, 1854; (*b*) (pt. 2) **4**:235, 1858.

18. Hansen, E.: Hospitalstid. **14**:1, 1871.

19. Schweigger, C.: Handbuch der speciellen Augenheilkunde, ed. 2, Berlin, A. Hirschwald, 1873, p. 456.

20. de Wecker, L., and de Jaeger, E.: Traité des maladies du fond de l'oeil et atlas d'ophthalmoscopie, Paris, A. Delahaye, 1870, p. 151.

21. Leber, T.: Ber. ü. d. Versamml. d. ophth. Gesellsch. **14**:18 and 165, 1882; Klin. Monatsbl. f. Augenh. **20**:18, 1882.

Who introduced ignipuncture in the treatment of retinal detachment? Martin,[22] de Wecker [23] and de Luca,[24] in 1881. After that time ignipuncture became generally accepted, particularly by French and English oculists, and was frequently employed.

Who was the first to select the region of the retinal tear and the place of the beginning of the detachment as the site for operation? Schoeler [25] of Berlin, in 1889. He injected tincture of iodine between the detached vitreous and the retina, in the neighborhood of the tear, and in the place of the beginning of the detachment; in other words, in the preretinal space. In his book he published an illustration from a case in which the retina became reapplied and the tear closed.

Who was the first to use ignipuncture at the site of the tear? Deutschmann, our senior master, in a case in 1896, which, however, he seems to have forgotten. The case was fully reported [26] in 1899. . . .

Who was the first to use ignipuncture systematically for a retinal tear and with success? Xavier Galezowski [27] of Paris, in 1902 and 1903, a few years before his death. He not only performed ignipuncture on the tear, but aspirated the retroretinal fluid before this procedure. He aspirated the fluid just as Gonin [28] did later, so that the retina could apply itself before the cauterization took place. "Je pratique l'aspiration du liquide et la galvanocautérisation de la partie déchirée de la rétine et de la choroide. J'ai obtenu ainsi de bons résultats." I practice aspiration of the fluid and galvanocauterization of the ruptured portion of the retina and of the choroid. In this manner I have obtained good results.[27b]

Larsson in 1932,[29] in discussing electroendothermy in the treatment of detachment of the retina, expressed himself so well that he is quoted here, in part:

> The hopelessness that used to characterize the ophthalmologist's attitude toward the question of treatment for retinal detachment has to some extent begun to lessen. Prospects have become brighter with the operative method of treatment suggested by Gonin. There is no doubt that ophthalmologists stand in great debt to Gonin for having so successfully and energetically taken up this question. A task of this nature becomes the more exacting as it involves, and will always involve, many great disappointments for the operator. Thanks to Gonin's work, the operative treatment for retinal detachment has become one of the vital issues in ophthalmology.
>
> Gonin's view of the pathogenesis of and operative treatment for detachment of the retina are already well known to every ophthalmologist. He maintains consistently de Wecker's old theory [published in 1870 [30]] of the importance of a rupture (hole) in the retina, as the primary factor in the origin of the detachment.

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22. Martin, G.: Tr. Internat. M. Cong. **3**:110, 1881.

23. de Wecker, L.: Ann. d'ocul. **87**:41, 1882.

24. de Luca, D.: Ann. di ottal. **12**:330, 1881.

25. Schoeler, H. L.: Zur operativen Behandlung und Heilung der Netzhautablösung, Berlin, Peters Verlag, 1889, pp. 28, 41, 86, 90 and 93; plate 1.

26. Deutschmann, R.: Beitr. z. Augenh. **40**:46, 1899.

27. Galezowski, X.: (*a*) Cong. de chir., Paris, 1902, p. 417; (*b*) Bull. et mém. Soc. franç. d'opht. **20**:214, 1903.

28. Gonin, J.: Rev. gén. d'opht. **37**:337, 1923.

29. Larsson, S.: Electro-Endothermy in Detachment of the Retina, Arch. Ophth. **7**:661 (May) 1932.

30. de Wecker and de Jaeger,[20] p. 153.

Gonin expressed the belief that if the hole can be made to close, the subretinal contents are absorbed and the detached retina resumes its normal position. He tried to achieve this closure of the hole by direct cauterization of its margins. Gonin [31] stated:

These unexpected successes were first referred to before the Swiss oculists at Bâle in 1919, with, of course, great reserve, then in some Swiss or French reviews, such as *Annales d'oculistique,* 1921, and *Revue générale d'ophthalmologie,* 1923, later, with a short description of some typical cases presented before the French, Swiss and German Societies in Brussels, Zurich and Heidelberg in 1925 and 1926. In 1927, there were shown at Berne several patients, the complete disappearance of whose detachments lasted for several months, and in the following year, at Lucerne, my colleagues, Siegrist of Bern and Vogt of Zurich, referred to their own confirmatory experiences; this was the first occasion on which credit was given to me by ophthalmologists other than my own fellow-workers and assistants. Later descriptions of cases or of the method were published in Italian, French or German in the reports of the ophthalmologic meetings held at Palermo, Paris and Heidelberg in 1928, and a more detailed account was published in the *Annales d'ocultistique* (May, 1930).

My actual experience on about 250 patients and in more than 300 operations can be summed up as follows:

1. In more than 95 per cent of the cases, whenever ophthalmoscopic examination is possible, one or several holes may be detected in the retina if looked for with sufficient care.

2. In about 10 per cent of these cases, the hole is not in the retinal tissue, but consists of a rupture or tearing away from its insertion at the ora serrata ("desinsertion of the retina").

3. In all recent cases, when the hole or tear has been closed, cure is immediate, complete and permanent.

4. In older cases (several weeks or months), closing the tear stops the detachment and may produce a more or less complete reposition of the retina, but restoration of the vision generally remains incomplete.

5. If the detachment relapses, it is found that the tear has not been completely closed or that there was another tear which had not been previously seen.

6. A recurrence of detachment in a different region of the eye is due to formation of a new hole in the retina.

It may appear curious that all my communications on that matter, numbering more than twenty, remained unknown to the English-speaking ophthalmologic world until the International Congress in Holland, no mention of them even being made either in an article in the *Lancet* [32] or in an article by Stallard [33] in the *British Journal of Ophthalmology.* Only following a recent visit to me at Lausanne by five members of the staff of the great eye hospitals in London were my ideas set forth in two papers published by Juler [34] and by Ormond.[35] The

31. Gonin, J.: The Treatment of Detached Retina by Searing the Retinal Tears, Arch. Ophth. **4**:621 (Nov.) 1930.

32. Operation for Detachment of the Retina, editorial, Lancet **2**:1202, 1929.

33. Stallard, H. B.: Brit. J. Ophth. **14**:1, 1930.

34. Juler, F.: Brit. J. Ophth. **14**:73, 1930.

35. Ormond, A. W.: Brit. M. J. **1**:940, 1930.

former described accurately the preliminary procedures [36] and the operation of thermopuncture; the latter dealt adequately with the anatomic side of the question.[37]

There is no doubt that a hole in the retina, a disinsertion of the retina at the ora serrata or a traumatic rupture of the retina play a tremendous part in (1) the causation of retinal separation, (2) the continuation of the separation and (3) the results of the operative closure, i. e., is wholly responsible for the success or failure of the operative treatment. At least one, and probably more, of these factors are involved in every case of retinal detachment observed for treatment. (These factors will be considered later in a more logical sequence.) They are mentioned at this time only to give further credit and honor to Gonin for his brilliant pioneer work. Since the advent of diathermy, chemical cauterization, superficial scleral electrocoagulation and catholysis, Gonin's thermocoagulation has been utilized in a steadily decreasing number of cases. It is to be remembered always, however, that his procedure was the first which could give the patient and the operator any assurance of a favorable result.

ETIOLOGY

In considering the etiology of retinal separation one must of necessity consider the mechanism of the separation. The two cannot be separated; together they comprise the pathogenesis. Further, it is impossible to divide retinal separations into a primary and a secondary classification, and even the recent subdivision of Troncoso,[38] who divided detachments into the idiopathic and the symptomatic type, and of Arruga,[39] who considered symptomatic detachment of the retina as

36. [Footnote quoted from the original article.] The whole procedure of locating the tear and preparing for the operation, as seen by Dr. de Jaeger, of Bruges, at Lausanne, has been again described by his partner, Dr. Rubbrecht, whose paper is summarized with full particulars and sketches by J. B. Lawford in the British Journal of Ophthalmology (**14**:359 [July] 1930). The only difference in Dr. Rubbrecht's method is that he makes a long scleral wound for the cauterization.

37. [Footnote quoted from the original article.] On a single point of importance, Ormond seems not to have realized my own conception of the changes in the vitreous. He stated that "liquefaction of the vitreous takes place." I do not think it does. I noticed only a separation of the vitreous fluid from its fibrillar mass, the latter being penetrated with epithelial cells and keeping thus a retractile strength sufficient to drag on the retina. I generally compare the retracted vitreous with a squeezed lemon, the cellular part of which has been not liquefied but simply deprived of its juice.

38. Troncoso, M. U.: Internal Diseases of the Eye and Atlas of Ophthalmoscopy, Philadelphia, F. A. Davis Company, 1937.

39. Arruga, H.: Detachment of the Retina, translated by R. Castroviejo, New York, B. Westermann, 1936.

belonging to a general group, with detachments caused by myopia, traumatism and chorioretinal conditions as other equally important subdivisions, are unsatisfactory and not correct. For this reason, such a classification will not be considered here.

When pathologically involved, the retina splits within itself, separating into two layers; the outer layer of pigment epithelium remains adherent to the choroid, while all the other layers are lifted from the pigment epithelium by serous exudates or hemorrhage or because of edema, cystic degeneration, neoplasms or cysts. Zeeman and Oltmanns [40] discussed in great detail this matter of trauma to the tissue and retinal separation. Their arguments and conclusions as to the mechanics of separation, based on cases of post-traumatic separation, are quoted in part:

> Structural cohesion is a function of the supporting tissue of the retina and also of the surrounding tissues so far as they are connected with the supporting tissue, that is to say, of the outer layers of the vitreous in its foremost part.
>
> The actual supporting tissue consists of Müller's fibers and *Spinnenzellen* (neuroglia cells). Müller's supporting fibers stand between the membrana limitans interna and the membrana limitans externa like so many columns which carry and hold together the retinal elements, together with numerous smaller glia cells, the thin fibrils of which encircle nerve fibers, ganglion cells and granular cells and dissolve into a kind of limiting layer (membrana limitans perivascularis) against the walls of the blood vessels (Krückmann). The broad ends of Müller's fibers, their cuticular thickened *Basalplatten* (basal plates), unite into a continuous limiting membrane, which extends into the cuticle of the ciliary epithelium. This layer, which by reason of its continuity resembles a boundary, a frontier between territories of different structure, can derive some strength from the solidity of its cellular construction. Yet we agree with Leber in considering the connection of the retinal elements translaterally looser than between its layers.
>
> Denig, with his experiments, not only refuted Berlin's idea that the picture of commotio retinae is connected with a subretinal hemorrhage but observed strange bumps reminiscent of a chain of pearls on the inner surface of the retina which made him presume that there were distention of Müller's fibers and small tears in the membrana limitans. Beginning detachment of this layer must, according to Lohmann, too, be the cause of the picture in cases of commotio retinae. Leber further pointed out that it has always been designated incorrectly as edema, that it is of no use to indicate the source which should produce the edema (in older theories, the vessels; in Denig's theory, the vitreous) and that in any case a swelling opacity is a much more suitable term. Microscopic tearing of the tissue elements, secretion of myelin, with its well known swelling capacity, a granular, intensely white opacity and rapid resorption are the bases of the picture.
>
> Differences in degree, greater distention of the retinal elements, wider tearing of the membrana limitans and tearing of the thickened outer layer of the vitreous create the conditions which, according to clinical observation, may later lead to the formation of a hole or to detachment.

Zeeman and Oltmanns expressed the belief that there are three definite facts of importance, i. e., (1) that the time which elapses between the onset of the lesion and the separation, which is its indirect conse-

40. Zeeman, W. P. C., and Oltmanns, H. J.: Trauma and Retinal Detachment, Arch. Ophth. **13**:971 (June) 1935.

quence, is variable and may be long; (2) that the retinal alterations which are first seen by the ophthalmoscope may be rather diffuse, spotted or cystic but hardly characteristic of changes universally considered as having been produced by trauma; and (3) that an eventual tear or separation at the ora serrata is not always the direct consequence of trauma. If the trauma is too light to cause commotio retinae, i. e., too slight to give rise to a beginning separation and thereby predispose to complete separation, it is impossible to assume that the separation which presents itself weeks or months later is due to this trauma.

As Zeeman and Oltmanns further stated, the relation of various traumatic and spontaneous forms of detachment makes it probable that the point of attack and the transference of the active forces (pathologic or physiologic) differ little if at all. The point of attack must lie in the supporting tissue of the retina. Only structures which are normally attached to the retina transfer forces to it and these are the anterior parts of the vitreous and the zonular fibers, that is, the very elements which play an important mechanical part in the physiologic accommodative mechanism. It is probable that the zonular fibers and the structures in the anterior parts of the vitreous show the lines of force and direction along which the active forces and the finer tissue shiftings connected with these active forces travel when the accommodative mechanism is brought into play, but these lines and forces must also be recognized as a menace to the retina, constructed as it is for some other function. There is an incongruity in the requirements seemingly made on the retina. The retina in a more limited sense, the sensitive derivative of the optic vesicle and part of the central nervous system, is in this instance passive; it does not bear but is borne; it scarcely resists but does not succumb. The skeleton of supporting tissue against and in the retina and along its vessels is in casu the important part in question here. This bears, resists, gives way and tears. It is not proved and is at least doubtful whether the Blessig grooves in the retina near the ora serrata may be called predisposing elements for tearing and detachment. This may be conjectured only so far as they are the consequence of, or are accompanied by, congenital or acquired weakness of the supporting elements, i. e., Müller's fibers and the membrana limitans interna, the glia coat and connective tissue along the vessels, the fibrous, thickened outer layer of the vitreous and the membrana hyaloidea posterior, and of the connections between these various elements. Coarser connections may be found among the innumerable more delicate elements. Tearing of the membrana hyaloidea and of the membrana limitans interna means tearing of the intraretinal supporting tissue and hence the first step in detachment of the retina. No wonder that the edges of the retinal tear often seem to lie in the normal retina even if there is a serious decrease in thickness in the immediate vicinity. Therefore, predisposing factors for complete separation appear to be all those factors

which cause in the delicate structure distention and fragility and thus promote the development of a tear in the membrana hyaloidea posterior. The reactive phenomena along the walls of the torn vitreous appear to confirm the great significance of this tear, as only rarely do they prevent such complications as retinitis proliferans or retinal and preretinal hemorrhages and never do they restore the retina.

Speciale-Cirincione [41] studied a series of enucleated eyes histologically to determine if possible the sequence of events which occurs in the eye with retinal separation. He described four stages. In the first stage the rods and cones are only slightly separated from the pigment epithelium, a number of processes from the rods and cones still being connected with the pigment epithelium but many being broken. No changes in the structure of the rods and cones are noticed. The vision in such an area may be as high as 1/10, and perception of color is present. In the second stage all the protoplasmic connections between the rods and cones and the layer of pigment epithelium are broken and massed together in a sort of membrane. This layer seems to pull the rods and cones in one direction at their external end. The vision in such an area is limited to perception of hand movements, and blue is the only color which can be distinguished. In the third stage swelling and degeneration of the rods and cones themselves become evident. The cones show more degeneration than the rods. The vision is limited to uncertain perception of light. In the fourth stage, observed in eyes with long-standing retinal detachment, no trace is left of the rods and cones, and marked degenerative changes are seen in the ganglion cells and other cells of the retina. The pigment epithelium in all these stages show two types of change, an irregularity in the arrangement of the cells and a tendency to proliferation through the subretinal space. Speciale-Cirincione expressed the belief that in the first and second stages, before changes in the rods and cones have occurred, a return of useful vision is possible if reattachment can be obtained.

Recently it was my privilege to study an eye microscopically [42] after a fairly successful reattachment had been obtained by means of the classic Guist-Lindner technic for trephining the sclera, with the subsequent application of potassium hydroxide. (The patient died in a state of diabetic coma three years after operation. She had been in a state of coma repeatedly for three months before her death, at which time she had preretinal, retinal and subretinal hemorrhages and complete hemiplegia on the left side.) The following quotations appeared in my previous report:

41. Speciale-Cirincione: Ann. di ottal. e clin. ocul. **53**:641, 1925.

42. Spaeth, E. B.: Reattached Retina: Physiologic, Ophthalmoscopic and Microscopic Observations and Comparisons, Arch. Ophth. **14**:715 (Nov.) 1935.

43. Footnote deleted by author.

Slow progressive retinal as well as choroidal degeneration was seen. Subretinal exudates and hemorrhages were present to outline the limits of the detachment—in some places to a marked degree, and in others in such slight amounts that one could just see a thin line of exudate; in some areas with, and in others without, early changes in the layer of rods and cones. A few less definite changes were seen in the epithelial cells. The layer of rods and cones seems to be the index of retinal viability. When these were not present as healthy looking elements, the other layers of the retina were also markedly impaired or even wholly wanting. The outer and inner nuclear layers when normal were rather certain to be accompanied by normal-appearing rods and cones. . . .

In the light of the observations just mentioned it is likely that the degree of recovery, in all its details, depends largely on the presence of healthy rods and cones and the absence of certain irregular subretinal cells, which probably are proliferated, epithelial cells. The observation of cholesterol crystals microscopically is almost certain proof that degeneration is so extensive that failure is certain. . . .

Anatomically, it is known that detachment develops between the outermost limits of the layer of rods and cones and the epithelial layer of the retina, i. e., in the layer of pigment epithelium. This region of separation is the space (if one can call it such) through which nourishment, by osmosis perhaps, normally reaches the outermost layers of the retina. It is possible that even in the presence of detachment these rods and cones continue to receive nourishment through this subretinal fluid, for in certain cases this fluid may be nothing but a transudate and pathologic only mechanically by reason of its presence. If it were pathologic in the inflammatory sense in all cases, the reason that recovery in some instances is so nearly complete, as observed grossly, is difficult to understand. At the same time, one can understand more readily the cause of the failures. Microscopic sections of detached retinas seem to show that as long as a certain number of filaments lying between the cells [see Zeeman, Oltmanns and Denig] of the pigment epithelium remain unbroken degenerative changes do not develop in the epithelium and in the layer of rods and cones. Only after loss of continuity has occurred do the multiple changes appear that are so commonly observed in detached retinas of long standing. It is perhaps through this maintained integrity of structure that recovery of function can occur after reattachment, even when detachment has existed for an indefinite length of time.

Arruga's and Troncoso's group of cases of primary, or idiopathic, detachment included cases of traumatism, myopia and chorioretinal diseases. They stated that the detachment in these cases should be called primary in that the retinal process was only distantly related to its original cause. Their group of cases of symptomatic, or secondary, detachment included cases of nephritic retinitis, acute uveitis, acute scleritis and orbital cellulitis; intraocular tumors and cysticercosis, and cardiovascular-renal conditions and diabetes. The detachment in these cases was considered secondary in that the development, course and prognosis of the separation was closely allied to an original and evident underlying basic disease. The manifest fault with this classification is that it considers these various types of detachment not as a symptom of the underlying disease (for this is what they are) but as the disease itself and pays only indirect attention to pathologic anatomy, that is, to

the underlying histopathologic factors. These include pathologic changes in the vitreous; disease of the sclera itself; disturbances in the equilibrium and the state of health of the intraocular fluids; disturbances in the relationship between intraocular tension and intravascular pressure; pathologic conditions of the choroid; retinal degeneration (retinosis), retinal exudates, retinal edema and retinal hemorrhages; and finally traumatic rupture and perforation in the choroid or in the retina. A consideration of retinal separation on the basis of idiopathic and symptomatic grounds may consistently include certain of these factors in any one type of separation, but it is equally certain that a histopathologic consideration, and that it is simply mechanistic, must be the basis for research and the study of the causes of retinal detachment. Success in the surgical treatment rests wholly within the realm, both in the possibilities and in the probabilities, of these anatomic principles. Gonin in 1919 stated that the large percentage of failures which occur in the treatment of this condition are due to the fact that the mechanism producing it is so poorly understood.

Anderson [10] considered four possible basic mechanisms, and into these four possibilities one may fit the various histopathologic probabilities. They are the theories of distention, of depression, of attraction and of exudation. Anderson's bibliography pertaining to these mechanisms is complete up to 1930.

According to the theory of distention, stretching of the choroid and the sclera is the actual cause of the detachment. Myopia, degeneration of the retina and trauma are all to be included in a consideration of this theory.

The theory of depression is the reverse of that of distention. A sudden loss of vitreous, atrophy of the eyeball, hydrostatic changes, ocular hypotension, detachment of the choroid, the hypotony of diabetic coma and the transudates and exudates of inflammatory processes are included in a consideration of this theory. Apparently these conditions modify or permit retinal separation by the withdrawal of a force which normally should assist in keeping the retina in position. It is doubtful whether hypotony of itself is the cause of separation. Certainly in most cases seen early in their course hypotony is not present, nor as Gonin [44] stated is it pronounced until many days or weeks from the time when the subretinal diffusion might be expected to stop. If the effusion plays the role of a compensatory exudation, the hypotony does not explain the limited character or the limited form of retinal tears. Moderate hypotony of itself is no contraindication to surgical intervention, and its disappearance after thermopuncture, electrocoagulation or catholysis is outstanding. This matter of effusion and of hypotony has been rather

44. Gonin, J.: Arch. d'opht. **51**:426, 1934.

well answered by a recent clinical case. After an uneventful corneoscleral trephining for simple noninflammatory glaucoma, a high degree of "choroidal" detachment developed. Sclerotic changes in the lens prevented any detailed observation of this, but visibility was adequate for diagnosis and for subsequent observation until complete recovery occurred. Some time later an equally uneventful intracapsular extraction was done. The ocular tension was constantly less than 12 mm. of mercury, both before and after extraction of the lens. After extraction one could see in the fundus at the site of the former "choroidal" detachment parallel curved lines of retinal striae outlining indelibly the position of former effusion in the presence of extreme hypotony. This case suggests an answer to the question.

The theory of attraction considers largely the possibilities of a pathologic process of the vitreous being the principal cause for the retinal separation. This theory, first considered by Hanssen [45] as an "attraction theory," is probably most intimately connected with the entire question of retinal tears, though the theory of distention has this same important relationship. The theory of distention undoubtedly explains retinal separation in myopic eyes and in those affected by trauma, with their frequent retinal tears, but it cannot explain the same condition in nonmyopic eyes and in eyes not affected by trauma. Müller,[46] Leber,[47] Lauber,[48] Gonin,[49] de Wecker,[50] Nordenson,[50] Heine,[51] Pillat,[52] Iwanoff,[53] and Lindner [54] are among the men who have felt that strands of vitreous, epithelial cell proliferation, the preretinitis of Leber, equatorial choroiditis, fibrillary degeneration, detachment of the vitreous, contraction of the vitreous and retinitis proliferans are likely causes.

The theory of exudation existed long before the present era of surgical correction for retinal separation. Leber stated that in the days before the invention of the ophthalmoscope "detachment" of the retina was recognized only after enucleation and was known [55] as subchoroidal

45. Hanssen, R.: Klin. Monatsbl. f. Augenh. **63**:295, 1919.

46. Müller, H.: Anat. Beitr. z. Ophth. **41**:363, 1858.

47. Leber, T.: Tr. Internat. M. Cong. **3**:15, 1881.

48. Lauber, H.: Klin. Monatsbl. f. Augenh. **72**:547, 1924.

49. Gonin, J.: Klin. Monatsbl. f. Augenh. **71**:232, 1923; **83**:667, 1929.

50. Nordenson, E.: Die Netzhautablösung, Wiesbaden, J. F. Bergmann, 1887, p. 4.

51. Heine, L.: Klin. Monatsbl. f. Augenh. **72**:305, 1924.

52. Pillat, A.: Klin. Monatsbl. f. Augenh. **69**:429, 1922; Ztschr. f. Augenh. **57**:347, 1925.

53. Iwanoff, A.: Arch. f. Ophth. **15**:1, 1869.

54. Lindner: Personal communication to the author, 1934.

55. Leber, in von Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1901-1911, vol. 7, pt. 2, chap. 10.

hydrops. The theory of exudation was originated by von Graefe,[56] who felt that serous choroiditis was the primary lesion in the majority of cases of spontaneous retinal separation. Detachment associated with intraretinal fluids, especially in those instances in which a fluid vitreous is present; with intraretinal hemorrhage; with nephritic chorioretinitis and nephritis; with the toxemia of pregnancy, and with inflammatory processes, such as iridocyclitis, tenonitis, tuberculous chorioretinitis and sympathetic ophthalmia, and the detachment which follows actinic retinitis are types which can be fitted within this classification. Experimental retinal separation is developed wholly on this basis. It is likely that the primary pathologic process in these instances reacts subsequently and unfavorably on the vitreous, causing degeneration of the vitreous and subsequently retinal separation through attraction or traction.

These four possibilities do not include the possibly mechanical separations due to cysts of the vitreous, choroidal and retinal neoplasms and retained intraocular foreign bodies. It is possible that any one, or perhaps all four, are relevant in these instances.

RETINAL TEARS

The presence of tears (holes) in the retina is a finding which is associated with practically all types of retinal separation, even those connected with inflammatory and exudative states. They may be subdivided into two general groups, disinsertions and all other tears. This subdivision is superficial and not especially satisfactory but is to be considered in that different types of etiologic factors are likely responsible for these two different types of tears.

A disinsertion is an oval or slit-shaped rent in the retina, occurring at the ora serrata and probably connected with cystic degeneration in the neighborhood of the ora serrata. At this point the retina separates from its pigment epithelium; i. e., there are no retinal elements, except the pigment epithelium lying peripheral to the rent. Traumatisms and traumatic rents in the retina often simulate these disinsertions. Disinsertions may be multiple, with the separated lobes lying close to each other. Two or more of these may unite as a larger rent, and in such instances a small island or flap of the peripheral portion of the retina can be found intact and still adherent to the ora serrata. These findings do not deny the statement just made; they simply indicate the process through which that disinsertion passed in its formation and in the formation of the peripheral flap still present.

This peripheral rent is often difficult to find ophthalmoscopically. The folds in the retina prevent a view of the peripheral portion of the retina, so that with even extreme dilatation of the pupil it may be impossible to view the neighborhood of the ora serrata. In every case

56. von Graefe, A.: Arch. f. Ophth. (pt. 2) **2**:202, 1855; footnote 17a.

in which the greatest elevation lies closest to the periphery one must suspect this type of tear. Further, when a most careful search of the fundus, frequently repeated, fails to show a hole in the retina at any place, then one should also consider the likelihood of a disinsertion being present. Changing the position of the patient's head and his body in bed or in a chair so that the retinal folds can fall away by gravity from the periphery will at times permit the operator to see these disinsertions. Preliminary subretinal drainage with a hypodermic syringe and needle is permissible in the case of larger bullous detachments and may also uncover a probable disinsertion. Pressure on the sclera while viewing the fundus as far peripherally as is possible has been suggested by Arruga as being of great assistance. Transillumination along the circle of the ora serrata has been definitely valuable in 3 cases which came under my observation, for it was the means of locating the disinsertion. Finding a disinsertion makes possible a much lessened amount of diathermic coagulation in that the rent can be completely sealed from the remaining portion of the retina, making unnecessary extensive treatment of the entire sclera overlying all of the separated retina, as would be almost obligatory in cases in which no disinsertion or other hole could be found.

The consideration of the remaining types of retinal tears is an interesting subject. The percentage of cases in which tears are found varies a great deal with the individual ophthalmologist. Arruga stated that in 1925 he found rents in 40 per cent of his cases of retinal detachment; in 1928, in from 60 to 65 per cent, and in 1932, in 80 per cent; at the present time he is finding them in from 85 to 95 per cent, depending on whether they are cases of old or of recent separation. Tears are most readily found in cases of recent separation in which the media are still transparent, but as the weeks pass after the onset of a retinal separation it becomes increasingly difficult to find them. Rest in bed, dehydration of the patient and subretinal drainage through scleral puncture are not infrequently necessary before the hole can be found. Multiple rents appear in from 25 to 30 per cent of cases. Because of this, the finding of a single hole should not halt further detailed search for additional tears. In the majority of instances the tears are located in the superior half of the retina, the temporal quadrant first in frequency, and the nasal quadrant second. The inferior temporal quadrant is third and the inferior nasal quadrant last in this order of incidence. This is diametrically different from the disinsertions, for the majority appear in the inferior periphery of the retina rather than in the superior periphery. The order of frequency from the periphery toward the macula is, first, between the ora serrata and the equator; second, in the macula itself, and, third, in that portion of the retina which lies between the macula and the equator. The form or the shape of these

retinal holes is as variable as are the innumerable potential probabilities. They may be many cornered, round or oval and arranged in groups or in rows or after the manner of perforations in coarse lace; they may be subdivided by an overlying retinal vessel or lying between the branches of a retinal vessel; they may be horseshoe shaped or crescent shaped, with the calks of the horseshoe or the horns of the crescent toward the periphery, and with or without hinged flaps; they may appear as large continent-like lacerations from trauma, involving sometimes an entire quadrant of the retina, or as slitlike holes, narrow and elongated, resembling hemorrhages and at times confused with hemorrhages; they may also occur as a concentrated group of tiny sievelike perforations. The macular holes are most commonly symmetric, while those at the equator are usually of a crescentic or a horseshoe shape. The region between the equator and the periphery presents the greatest variety of holes, while at the extreme periphery holes and disinsertions again assume a more regular outline, being large and spindle shaped, with their long axes parallel to the curve of the ora serrata. Traumatic

TABLE 1.—*Incidence of Various Types of Holes in Relation to Refractive Error*

	High Myopes	Low Myopes	Emmetropes	Aphakics
Disinsertion	3	6	27	1
Round	15	8	6	1
Arrow-head and horse-shoe	12	7	4	..
Slit	2	2	1	..
Irregular	..	1	3	1

lacerations are of all varieties and shapes and usually are the largest in size. Not commonly, but still not rarely, one may find both disinsertions and extraperipheral tears present in the same case. Extramacular and macular tears may also be present simultaneously; this is more rare but has been observed.

Shapland's [57] classification of the holes in 100 cases is quite a satisfactory one:

(1) Festoon-shaped, situated at the extreme periphery, and corresponding to Prof. Gonin's "disinsertion" at the ora serrata.

(2) Rounded, punched-out holes, suggesting the possibility of a pre-existing cystic degeneration of the affected portion of the retina, or an origin in a previous focus of choroiditis.

(3) Arrow-head and horse-shoe-shaped rents, in which the point of the arrow or the convexity of the horse-shoe are always on the disc or central side of the base of the arrow-head or concavity of the horse-shoe.

(4) Radial slit-like tears. It is worth pointing out that the limbs of a horse-shoe rent may be mistaken for two radial tears, the convexity of the horse-shoe being hidden behind a secondary fold of detachment.

(5) Irregular rents.

57. Shapland, C.: Tr. Ophth. Soc. U. Kingdom **51**:152, 1931.

In Shapland's series there were 37 cases of the first type, 30 of the second type, 23 of the third type and 5 each of the fourth and fifth types. Table 1 (Shapland's) shows the incidence of these various holes as connected with the refractive error.

Gifford,[58] before Gonin's death (which occurred June 10, 1935), questioned him (Gonin) as to the success he had had in cases in which the holes were large. Gonin's picturesque words follow verbatim:

> C'est plus facile de tirer sur un elephant que sur un lapin, mais pas si facile de le tuer (It is easier to shoot an elephant than a rabbit, but not as easy to kill him).

THE MECHANICS OF THE FORMATION OF RETINAL TEARS

A consideration of the mechanics of the formation of retinal tears includes a consideration of two basic factors, the architecture of the eyeball and the various structures related to it and the strong probability of different development of macular and extramacular tears. The explanation of the influence of the first of these factors is based on the relationship which exists between the rigid sclera, the inelasticity of the choroid and Salzmann's observation that spontaneous tears occur in the lamina vitrea, not involving the choriocapillaris. Anderson stated that Parsons concurs in this. The pressure of the various rectus and oblique muscles on the eyeballs may be a factor in changing intraocular pressure because of occlusion of the vortex veins. This would be an example of an increase of intraocular pressure due to physiologic pressure as outlined by Edridge-Green and again referred to by Anderson. Next is the consideration of the vitreous itself so far as retinal separation is concerned; the vitreous must be classed as a tissue subject to the changes that all tissues are subject to, and by its intrinsic attachments to the retina itself it will affect the retina secondarily when undergoing edema, shrinkage and inflammation or when lost, though only in part, through operation or by trauma. The last two factors are also considered by Comberg [59] and by Levinsohn.[60] Comberg has shown that there is a vibration of the posterior portion of the eye when one is reading and that the eyeballs are pushed backward when the lids are closed for an amount equal to from 15 to 20 Gm. in weight. Levinsohn considered that the position of the head and the effect of gravity are most important. He developed myopia experimentally in monkey's eyes by keeping their heads downward and concluded thereby that the abuse of the eyes in ordinary life is a main factor in the development of this, rather than heredity, this being only one of several other determining factors.

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58. Gifford, S. R.: Surgical Treatment of Retinal Detachment, Arch. Ophth. **16**:405 (Sept.) 1936.

59. Comberg, W.: Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. (1928) **47**:126, 1929.

60. Levinsohn, G.: Arch. f. Augenh. **102**:308, 1929.

In addition to these factors, further conditions may be relevant, so far as macular holes are concerned. The effects of direct trauma, especially when applied to the anterior segment of the eye, is significant. Tears produced in this manner are almost always round or oval, while the more frequent peripheral tears are angular, crescent shaped and slitlike, being ragged and usually large. It seems as if the macular holes due to trauma suggest an explosive effect, as if increased pressure in the fluids within the retina was released forward, toward and at the macula. Also, a more intimate anatomic relationship which exists at the macula between the retina and the choroid is undoubtedly a further factor.

There is no doubt that a definite relationship exists between retinal holes and retinal separation. The latter is dependent wholly on the first, and without the development of the first, forces which would otherwise cause retinal separation will be expended and their effects come to naught; i. e., while a potential separation is in process, spontaneous recovery occurs from these potentialities because a tear does not develop. In supporting Leber's statement that sudden retinal detachment could not occur without a hole, Lister [61] stated:

> With an intact eyeball and an intact retina, no detachment can take place unless there is a simultaneous and practically corresponding outpouring of fluid into the inter-retinal space on the one hand and contraction of the vitreous on the other. Such pouring out of inter-retinal fluid and absorption of the vitreous cannot, of course, take place suddenly. The onset of any separation of the retina without a hole must, therefore, of necessity be gradual. It would, therefore, appear that if it can be substantiated, in any given case, that the loss of sight from separation was not merely suddenly detected but actually occurred suddenly, we can definitely predict the presence of a hole, whether or not it can be seen by the ophthalmoscope.

According to Anderson, one can conclude that the majority of detachments which arise suddenly are associated with the formation of a hole but not that the relationship is essential and absolute. In the light of more recent surgical methods and the more recent statistics, I believe that this relationship is "essential and absolute."

A retinal tear from an adherent contracting vitreous, with the tear and separation starting above, will permit subretinal fluid to infiltrate from above with but a minimal separation there (above); however, there will be a maximum of separation below. This is a finding seen not uncommonly. In these instances infiltration of vitreous through the tear is not as important as is that of the subretinal fluid. An area of preretinitis will cause adherent bands in the vitreous to the vitreous surface of the retina, resulting in a hole with an initial minimal separation. The separation then will continue and will grow owing to the passage of vitreous through this hole as a result of constant ocular

61. Lister, W.: Brit. J. Ophth. **8**:1 and 305, 1924.

rotations and unceasing rotary shocks to the vitreous body, on account of its inertia, as first outlined by Lindner, and not because of any continued traction on the retina. This force was expended when the retinal tear occurred, and the damage is continued by subretinal prolapse of the vitreous. In the formation of the common crescent-shaped and horseshoe-shaped holes, the hole always lies on the distal side of a fold with the arms of the tear toward the ora serrata because the pedicle of the fold or flap remains attached to the retina near to the ora serrata.

The accompanying illustration is a composite drawing of an actual instance of retinal separation with a small single hole at the macula. There were no other tears, rents or disinsertions present. The other holes illustrated have been sketched in at the approximate positions where they have actually appeared in other patients, as closely as this sketch would permit. The drawing thus shows some of the various common rents found.

A and *K* are the ordinary horseshoe-shaped tears mentioned previously. *B* is a small peripheral traumatic (direct blow to the eyeball) rent. *D* is a peculiar shaped peripheral tear with two flaps, the larger of the two flaps being folded in part on itself and the smaller lying free, a bridge passed over this tear continuing along a large retinal vein. *E* and *H* are two linear tears which appeared on the dome of bullous separations. *H* was undoubtedly a rent at an area of choroiditis. *E*, following operation and the reduction of the separation, turned out to be a considerably larger tear, bilobed with the larger lobe hidden underneath the fold. *I* and *L* were the two extremes of a tear which lay at the extreme ora serrata, which certainly resulted from peripheral cystic degeneration of the retina. *J* is a tear with a wrinkled flap, similar to one which Arruga demonstrated in his case. *M* and *N* show the appearance of the usual retinal disinsertion.

When one considers, therefore, the scleral elongation produced by myopia and the pathologic changes in the vitreous in myopic eyes; the many possible structural changes due to chorioretinitis; Arruga's pertinent observation that the retina does not detach, when it is adherent to both the vitreous and the choroid, as in dissemiated choroiditis, while equatorial and peripheral chorioretinitis frequently accompanies separation of the retina; the various atrophic and cystic degenerations which occur in the retina; the possible structural changes in the retina, which are of a developmental nature (considered highly important by Vogt); adhesions between the vitreous and the retina; the multiple minimal traumatisms to which the eyes are constantly subjected, and the elasticity of a ruptured pathologic retina, then only is one aware of the factors concerned in the development of retinal rents. When one adds to these certain conditions practically unknown, such as disturbances

of intraocular tension, intravascular pressure or fluid equilibrium and the effects which the albumins of the vitreous and the water of semi-fluid vitreous may have on the retina and choroid, one should not be astonished at the number of retinal separations which occur. Conversely, one must agree to the absolute relationship which the tear plays to retinal separation and, even more, express sincere astonishment that such separations are relatively infrequent.

The relationship which retinitis plays to retinal separation, either from direct damage or because of a response to the toxic effects of tissue damage, was shown rather well by Harman and MacDonald [62] in their paper on detachment of the retina due to exposure of light dur-

Composite drawing of a retinal separation with a small hole at the macula. (The letters are explained in the text.)

ing an eclipse. The eye showed an extensive separation of the retina involving the whole of the upper half of the eye, the separation being ballooned downward so as to obscure the macula and the disk. The sequence of events in their case seemed to be as follows: (1) severe damage to the macula, consequent to exposure to sunlight; (2) a succeeding infiltration of the macular region; (3) extension of the exudate in such a quantity as to cause a gross separation of the retina; (4) absorption of the greater quantity of the fluid during the weeks of rest; (5) exposure (uncovering) of the initial area of damage in the macula, and (6) onset of scarring in the macular region.

62. Harman, N. B., and MacDonald, P.: Brit. M. J. **1**:637, 1922.

Knapp[63] reviewed Van Manen's analysis of 124 cases in which operation was performed during 1935 at the Weve clinic in Utrecht. There were 31 failures in the series. Ten of these must be deducted for sufficient reasons.

The author arranged the cases into groups for a survey of the results. Group 1 consisted of cases of retinal separation in aphakic eyes. This group was further subdivided into two groups: cases in which detachment occurred in an aphakic eye and those in which the detachment preceded the extraction, requiring that the cataract first be extracted. In the first group 6 operations were performed with success in 4; in the second, 7 operations with success in 1. Group 2 was composed of 4 cases of retinal detachment which followed a perforating trauma; cure was effected in 2. Group 3 consisted of 3 cases of detachment with a tear in the fovea; cure was obtained in all. (Though improvement in vision occurs, coagulation in the macular region prevents the return of good vision, and a central scotoma is always present.) Group 4 consisted of cases of retinal detachment in myopic eyes with giant tears; 4 operations were performed, 3 of which were successful. Group 5 was composed of 20 cases of retinal detachment with a tear at the ora serrata; recovery occurred in all. Group 6 consisted of 32 cases of detachment with the larger part of the separation situated above the horizontal meridian; successful results were obtained in 26. (The treatment in these cases was the same as that usually given, except that the degenerative areas surrounding the tears or suspicious areas in any other meridian were, in addition, treated by surface coagulation.) In group 7, cases of retinal detachment in which the larger part of the detachment was situated below the horizontal meridian, 25 operations were performed with success in 20. Group 8 consisted of cases of detachment with centrally situated folds in the retina; 8 operations were performed with success in 2. In group 9, cases of nonidiopathic separation and retinitis proliferans, 4 operations were performed with failure in 3.

The summarization of two series of cases, that of Baer and Shipman,[64] and that of Dunnington and Macnie,[65] is presented in table 2. This table illustrates certain salient and interesting relationships. (It is remarkable to note the close similarity in percentages in many of the comparisons.) These data are presented not from a therapeutic standpoint but to show and emphasize common etiologic factors and the factual relationships such as sex, age, retinal tears and myopia.

63. Knapp, A.: Die diathermische Behandlung der Netzhautablösung in der Universitätsaugenklinik Utrecht und ihre Ergebnisse im Jahre 1935, by J. G. Van Manen, Book Reviews, Arch. Ophth. **17**:387 (Feb.) 1937.

64. Baer, B. F., Jr., and Shipman, J. S.: J. M. Soc. New Jersey **34**:494, 1937.

65. Dunnington, J. H., and Macnie, J. P.: Detachment of the Retina: Operative Results in One Hundred Fifty Cases, Arch. Ophth. **13**:191 (Feb.) 1935.

RETINAL SEPARATIONS FROM DIRECT TRAUMATISM

The force of a traumatism need not be one, necessarily, of great degree; in fact, injuries from objects such as soft balls, from a bump on the back of the head or from a sudden jar in missing a step are as frequent an etiologic factor as are injuries from a blow with a fist, from sticks, elbows or stones, from a sharp edge of a door or from the back of a chair. These are actual injuries which occurred in various cases. Perforating injuries are somewhat different in effect. In such cases the choroid is also ruptured, perhaps even more extensively, so that the hole develops suddenly and abruptly. Subchoroidal and subretinal hemorrhages appear as precipitously and the

TABLE 2.—*Summary of the Data on Two Series of Cases of Retinal Separation*

	Baer and Shipman 44	Dunnington and Macnie 150 (155 eyes)
Total number of cases reported		
Females	31.8%	36.0%
Males	68.2%	64.0%
Cases in which myopia of varying degree was the refractive error	50.0%	61.9%
Cases in which 2 operations were necessary and done	27.2%	16.4%
Cases in which 3 operations were necessary and done	4.5%	4.5%
Cases in which retinal tears were seen	40.9%	56.0%
Successful results in cases in which tears were seen	55.5%	41.8%
Successful results in cases in which tears were not seen	38.4%	34.7%
Cases with history of injury	29.6%*	41.3%
Successful results in cases following injury	46.1%†	36.0%‡
Total cases with no improvement in the field of vision and continued detachment of the retina	38.6%	51.7%
Total cases with slight improvement (partial detachment of retina; slight improvement in visual field and central vision)	15.9%	13.7%
Total of successful results (retina in place; full field; improvement in central vision)	45.5%	34.3%
Age of youngest patient operated on	16 yr.	5 yr.
Age of oldest patient operated on	79 yr.	75 yr.
Average age	45.5 yr.	39.8 yr.

* This group included 5 cases of aphakia.
† This group included 5 cases of aphakia, in only 1 of which operation was performed.
‡ This group included 12 cases of aphakia, with cure in 3; 41.6 per cent of the patients had hyperopia and 27.4 per cent myopia.

vitreous is injured simultaneously. Because of these three factors the separations may occur immediately, or several days or weeks later or even after a delay of several months. Further, in all traumatic separations one must not forget that while the trauma may be the real cause of the separation, some of these eyes may have been affected prior to the injury by undiagnosed forms of chorioretinitis or iridocyclitis. One such instance is especially outstanding. A young man had complained of impaired vision several weeks before a fist fight. His attending physician, not an ophthalmologist, had examined the fundus himself and had informed the boy that the lower half of his retina was studded with signet-shaped rings of pigment. Twenty-four hours after a fight in which he was struck in the eye the entire inferior portion of the retina separated, with a massive rent running from 9 o'clock around to 4, gaping so widely in the inferior nasal quadrant that

the choroid was entirely visible in this region. The peripheral lip of this rent was invisible because of a dense crescentic-shaped hemorrhage which extended from the lip of the rent to the ora serrata. (Surgical treatment was unsuccessful in this case.)

The mechanism of traumatic separations is perhaps the most readily understood of all etiologic factors, even though possibilities are based wholly on theory. The eyeball as it rests in the orbit is subject to posterior impact from the bony walls of the orbit, because of the resistance of the orbit to transient flattening of the globe from a blow; because of its position, the majority of traumatisms will be applied from the front and perhaps slightly downward. Repeatedly, retinal separation seems to be precipitated by minor traumatisms of such inconsequential force and so distantly applied that their significance is often lost and not detailed by the patient when his history is taken. Because of the resistant bony orbital wall, retinal and choroidal rupture are not uncommon by contrecoup. In these instances the tear is usually at the posterior pole about the papilla or in the macula, and the choroid alone may be lacerated, the overlying retina remaining intact. The common findings in the macula in cases of contusio bulbi and of commotio retinae seem to indicate the delicacy of the retina at this point and the effect of countrecoup. The temporary deformity or flattening of the eyeball due to a blow probably accounts for the large irregular giant ruptures which occur in the region of the equator. The edges of the laceration are turned inward, i. e., away from the sclera, and with the ophthalmoscope one can see that the enormous force, suddenly applied, plus the transient momentary deformity of the scleral shell, has resulted in an explosive rupture of the choroid and the retina together or singly. Giant traumatic retinal lacerations may be due not to the abrupt infolding but to a sudden stretching from the impact to, and the subsequent deformity of, the scleral shell. While disinsertions are probably due to peripheral cystic degeneration of the retina or to peripheral degenerative chorioretinitis, traumatic tears which appear in the preequatorial zone may simulate disinsertions. They probably are not true disinsertions but actual rents through a portion of the retina which was already thin and atrophic by reason of this peripheral retinal pathologic process. Choroidal and retinal ruptures, which occurred so commonly during the World War as a result of bullet wounds in the neighborhood of the eyeball (without actual perforation of the eyeball), were undoubtedly of this same explosive nature. These showed the ruptures of contrecoup, ruptures through the posterior pole and irregular and often huge ruptures close to the path of the missile through the bony orbit. Perforations of the sclera may result in destruction of the globe, especially if the force of impact has been great or the missile large. Stab wounds, with the immediate withdrawal of the penetrating weapon, and smaller perforating wounds, with probably retained

foreign bodies, cause retinal tears, first, by perforation; second, by the loss of some vitreous; third, by the retraction of the lips of the wounds by contraction; fourth, by the continued gaping of these wounds by the deposition of scar tissue in the vitreous and in the path of the missile, with consequent drag on the retina, and, fifth, because of the hemorrhage connected with the original wound and the slow absorption so common of hemorrhages into the vitreous. The frequency of retinal separation following extraction of magnetic foreign bodies by the posterior route is significant.

It has been a frequent observation that retinal separation follows late after retinitis proliferans. The injuries received during the World War seemed to show on analysis that choroidal rupture, even though of necessity complicated by a lacerated overlying retina, was followed in relatively few instances by retinal separation, either immediate or late. This observation is not consistent with that just mentioned relative to the choroidal and retinal damage connected with extraction of a foreign body by the posterior route. Massive retinal, subretinal and preretinal hemorrhages alone, as seen in pregnancy, Hodgkin's disease and diabetes, do not of themselves cause retinal separation. A tear from a contusion, however, plus a lesser degree of hemorrhage, will do this, and more frequently than when the choroid has also been ruptured by the concussion of the contusion. It seems as if the accompanying choroidal damage protects that patient from a later retinal separation.

MYOPIA

In from 50 to 75 per cent of cases of retinal separation myopia seems to be a major predisposing factor. Racial factors peculiar to an environment may change this, but the probable average is in the neighborhood of 60 per cent of all cases. The mechanism of the retinal separation in cases of myopia, according to Majewski [66] in a consideration of von Graefe's original opinion, demands that the myopia itself be due to posterior sclerochoroiditis and that the subsequent retinal separation (as described by Arlt, by Leber and by Nordenson) presupposes the development of an inflammatory process in the choroid consequent to the sclerochoroiditis, with a resulting serous exudation. This pathologic subretinal condition, added to an increase in the size of the globe (which can be considered approximately spherical), should be the anatomic basis for separation in cases of myopia. The sequence, however, which exists between the tear and the separation, or inversely, between the separation and the tear, is still unsolved. Certainly separation and choroiditis is an established entity; in fact, Elschnig [67] in 1931 presented a

66. Majewski, K. W.: Arch. d'opht. **47**:273, 1930.

67. Elschnig, H. H.: Klin. Monatsbl. f. Augenh. **86**:595, 1931.

case of primary choroidal detachment of unknown etiology in which there was a humplike, rigid and sharply demarcated mass of the usual fundus color of choroidal detachment and in conjunction with this, a retinal separation which included more than the lower half of the retina. Recovery was uneventful, with vision of 6/8 following subretinal drainage through a scleral window. Anderson stated:

> . . . as the sclera and the choroid stretch, the normal fine relations between the retinal pigment epithelium and rods and cones are broken down. The vitreous becomes degenerated and the retina atrophic. This eye is now susceptible to toxic influences or circulatory disturbances make for effusion hemorrhage, and to trauma or exertion which can easily cause rents or apertures in the degenerate retina.

Both de Long and Samuels (as expressed at various times in their work on peripheral cystic degeneration of the retina) expressed the belief that while this peripheral cystic degeneration may be peculiar to myopia, it is not the cause, but the result, of the myopia; further, retinal separation is due to this cystic degeneration and not to the elongated scleral shell of myopia or even to the stretching of the retina (if this occurs)—a fine distinction but a necessary one.

INFLAMMATORY CAUSES [68]

Chorioretinitis; cystic and senile degeneration of the retina; edema of the retina, as seen in association with nephritis, and peripheral chorioretinitis, uveitis and scleritis are all conditions characterized by two types of pathologic change, degeneration and atrophy. It is probable in these instances that the mechanism of retinal separation is similar to that of the separation associated with myopia. Certainly a tear must initiate the condition, and histologic sections have been described wherein patches of chorioretinitis were the exact site of a retinal rent. The central hole in the macula following contusio bulbi and the similar holes which follow actinic retinitis seem to prove this sequence of events conclusively. It is not certain whether tuberculous chorioretinitis and syphilitic retinitis are the cause of retinal separation. Conjectures may be made, but as yet no definite proof is present. Diabetic retinitis, excluding subretinal hemorrhages, if not nephritic in nature does seem to be a cause, but here also in individual cases other nondiabetic factors could be the true underlying reason for the separation.

SYMPTOMATIC SEPARATION

Retinal separation which occurs with sarcoma of the retina and of the choroid and with cysticercosis and hydatid cysts is purely symptomatic and is of interest only in reaching a correct differential diagnosis.

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68. See "Myopia."

APHAKIA AND RETINAL SEPARATION

The retinal separation following early after operation for cataract is almost a distinct and definite type. Certainly preexisting retinal conditions play a part, for it has been known for years that the myopic eye is prone to retinal separation following the extraction of cataract. Cases of this type are to be considered probably and essentially as instances of separation due to myopia, and operative treatment should be employed the same as for any other detachment not basically traumatic. The elongated scleral shell, the preexisting myopic pathologic process, the probable development of a larger optically empty space behind the contracted vitreous, anterior to the retina (because of the trauma and the structural changes following extraction of cataract), the probability of a fluid vitreous and the possibility of the loss of some of this are the physiologic and structural factors responsible for this form of detachment. Of all factors, these are least commonly associated with a demonstrable retinal tear. For this reason it is likely, though not proved, that many of these tears are the result of uncovered retinal disinsertions. Further, they occur shortly after the extraction of cataract and seldom if ever late; in fact, a late development would place them in the second class of postoperative (cataract) separations.

This second group of postoperative separations include those instances which follow late after the extraction of cataract and after discissions, sometimes single but more often multiple; they are occasionally seen after linear extractions for traumatic cataracts and no doubt play a part in those cases in which the patient has a stormy postoperative convalescence with a more or less long drawn out iridocyclitis.

The etiologic factors in the first group of postoperative separations are probably connected with Anderson's theories of distention and perhaps also of depression. This second group, however, is probably, with the separations due to trauma, the best example for demonstrating the theory of attraction. The operation which antedated this second group has resulted in a demonstrable contraction of the vitreous, with cicatrices within the vitreous body and chamber. In 3 instances a postoperative separation has been correctly prophesied following earlier operation of this type simply because one could see with the slit lamp a contracting vitreous with tension lines. These arose in the neighborhood of the temporal portion of the ora serrata above and diverged downward and backward, like the veins of a palmleaf fan. In 2 instances rents were found in the retina, but only after the separation had been found. In 1 instance the separation occurred during an interval of five days and in the second, within a period of thirty-six days. In neither of these was operation successful. The third instance was discovered suddenly by the patient, though it probably was present for from a week to ten days.

The patient had had a linear extraction for traumatic cataract, a disinsertion was found, and operation was successful in completely reattaching the retina.

These separations are considered hopeless by many operators. This is an unduly pessimistic attitude. Etiologically, they differ so little from any of the other types of separation that all similar rules for surgical treatment apply.

CONCLUSIONS

1. The presence of a retinal tear or of a disinsertion is necessary to the development of retinal separation.

2. Histologically, the theories of distention, of depression, of attraction and of exudation, play equally important roles in the causation of retinal separation, though under varying circumstances and peculiar to individual cases.

3. Myopia itself may not be the etiologic cause but the retinal changes (retinosis), degenerative and trophic, which result from myopia.

4. The retinal separation in aphakic eyes is caused by the same factors which are responsible for retinal separation in nonaphakic eyes.

5. The retinal separation of directly applied traumatisms is probably to be explained by the theory of distention plus the damage to the ocular tissues from the impact, the deformity-producing blow (affecting the shape of the eyeball) to be considered as distinct from the damage to the tissues themselves from the impact. The laws governing the transmission of energy through a complex fluid body, as is the eyeball and its contents, apply as rigidly here as with any less complex mass of fluid or semifluid.

1930 Chestnut Street.

News and Notes

SOCIETY NEWS

New York Academy of Medicine.—At the November meeting of the Section of Ophthalmology of the New York Academy of Medicine, held November 21, an instructional hour on perimetry of chiasmal lesions was conducted by Dr. Ralph I. Lloyd. Slit lamp demonstrations were made by Drs. Milton Berliner, Wendell L. Hughes, Giralamo Bonaccolto and Gordon M. Bruce.

Dr. Mark Schoenberg reported a case of recurrence of ocular hypertension eighteen years after an Elliot trephining.

Dr. Phillips Thygeson gave a report on the use of sulfanilamide in the treatment of inclusion conjunctivitis.

A paper on the use of the grenz ray in the practice of ophthalmology was read by Dr. Raymond Pfeiffer and discussed by Dr. Gustave Bucky.

The paper of the evening was presented by Dr. Walter Ivan Lillie, of Philadelphia, on the interpretation of fundal changes associated with arterial hypertension.

Dr. James W. White is chairman of the section and Dr. Rudolph Aebli, secretary.

Swiss Ophthalmological Society.—At the annual meeting of the Swiss Ophthalmological Society in Zurich, Professor Vogt presented the society with 50,000 francs to celebrate the thirtieth anniversary of the society and in memory of his son, Alfred Vogt, who died ten years ago. The fund is to be known as the Alfred Vogt Foundation, and the income is to be used every year in the form of a prize for the best scientific contribution by a member of the society and to facilitate scientific publications.

Ophthalmological Society of the United Kingdom.—The annual congress of the Ophthalmological Society of the United Kingdom will be held at the headquarters of the Royal Society of Medicine, Wimpole Street, London, W. 1, on April 20-22, 1939. The subject for discussion will be "The Problems of Refraction." The discussion will be opened by Mr. Affleck Greeves, Mr. Charles Goulden and Mr. A. Harold Levy.

The Bowman Lecture will be delivered by Professor Weve, his subject being "Diathermy in Ophthalmic Practice."

The International Organization Against Trachoma and the International Association for the Prevention of Blindness will hold their annual meetings on Wednesday, April 19, 1939, at the same venue as that of the Ophthalmological Society of the United Kingdom. These meetings will be accessible to members of the congress.

The annual dinner of the society will be held on Thursday, April 20. The president of the society is T. Harrison Butler Esq., M. A., D. M.

National Society for Prevention of Blindness.—The annual meeting of the National Society for the Prevention of Blindness was held on

Thursday, December 1, at the Russell Sage Foundation Building in New York. The following addresses were given: "Thirty Years in Saving Sight," by Lewis H. Carris, and "Prevention of Blindness from the Ophthalmologist's Point of View," by Dr. Ellice M. Alger. The presentation of the Leslie Dana Gold Medal to Dr. Ellice M. Alger was made by Dr. John N. Evans.

First de Schweinitz Lecture.—Dr. Edward Jackson, of Denver, delivered the first de Schweinitz lecture of the Section on Ophthalmology of the College of Physicians of Philadelphia on November 17 on "The Development of Ophthalmology in One Lifetime." The lecture was established in honor of Dr. George E. de Schweinitz, who died on August 22.

General News

Archiv für Augenheilkunde.—The *Archiv für Augenheilkunde,* at the conclusion of its one hundred and tenth volume in November 1937, was combined with the von Graefe's *Archiv für Ophthalmologie.* The new journal carries on its frontispiece the following title: "Albrecht von Graefe's Archiv für Ophthalmologie, vereinigt mit Archiv für Augenheilkunde. Redigiert von A. Wagenmann, Heidelberg; W. Löhlein, Berlin, and E. Hertel, Leipzig."

This means the termination of the *Archiv für Augenheilkunde* as an independent journal. This journal was founded in 1869 by Herman Knapp (New York) and Moos (Heidelberg) as the *Archives of Ophthalmology and Otology* and as the *Archiv für Augenheilkunde und Ohrenheilkunde.* It appeared in English and German editions up to 1911. In 1879 the journal was divided into two journals, the *Archives of Ophthalmology* and the *Archives of Otology,* and the *Archiv für Augenheilkunde* and the *Zeitschrift für Ohrenheilkunde.* The ophthalmic journal was edited by Herman Knapp and Hirschberg (Berlin). In 1882 Schweigger (Berlin) took Hirschberg's place as the German editor; in 1905 Hess (Munich) became editor; he held this position until his death in 1925, when Wessely (Munich) took his place and was later joined by Hertel (Leipzig). An important part of the *Archiv für Augenheilkunde* was the review of the literature. This was abandoned in 1925, when the *Zentralbalt für die gesamte Ophthalmologie* was founded.

The *Archives of Ophthalmology* was continued by Arnold Knapp and W. A. Holden from 1912 until 1928, when the *Archives* was taken over by the American Medical Association, with the following editorial board: Arnold Knapp, chief editor; G. S. Derby; W. Zentmayer; S. R. Gifford; F. H. Adler and J. H. Waite.

New Editors for Zeitschrift für Augenheilkunde.—A change has taken place in the editorship of the *Zeitschrift für Augenheilkunde,* which has been conducted, with such well known success by G. Behr (Hamburg) and J. Meller (Vienna). The names of A. Brückner (Basel) and H. Weve (Utrecht) have been added to the list of editors, and it is reported that the latter two will be the chief editors of the journal beginning with January 1939, when certain changes in the publication are being considered. The high standing of the *Zeitschrift*

für Augenheilkunde has been due to the indefatigable work of J. Meller, and it is to be hoped that for the future success of the journal his cooperation will not be lost.

New Ophthalmic Journal.—Welcome is extended to a new bimonthly ophthalmic journal, *Archivos brasileiros de oftalmologia.* The articles will appear in Portuguese, French, Spanish and Italian, with a résumé in Portuguese. The editors are Drs. W. Bedford Mattos, S. Paula Santos and Durval Prado, and the address of the publication office is Caixa Postal 4086, São Paulo, Brazil.

Acta Ophthalmologica Oriental.—Dr. Aryeh Feigenbaum writes that he and his whole staff of collaborators have withdrawn from the editorship of the *Folia ophthalmologica orientalia,* founded and edited by him for a number of years, and that he has founded a new journal, the *Acta ophthalmologica orientalia,* of which the first issue has just appeared.

UNIVERSITY NEWS

Course in Basic Ophthalmology.—A new course in basic ophthalmology began November 1 at Wayne University College of Medicine, Detroit, to continue each Tuesday and Friday throughout the year. Designed for residents and those interested in increasing their basic knowledge in ophthalmology, the first course is on histology, comparative and human, while the second will be on physiologic optics, physiology of the eye, and gross anatomic sections of the head and neck. There is no fee for the courses. Dr. Parker Heath is professor and head of the department of ophthalmology.

Postgraduate Course in Biomicroscopy.—A one week course for licensed physicians specializing in diseases of the eye will be given at Wayne University College of Medicine, Detroit, December 12 to 17 inclusive, under the direction of Dr. Parker Heath, professor of ophthalmology. Gullstrand's principles and their modifications will be discussed. Practical training in the use of various instruments for slit lamp microscopy will be emphasized. The class will be limited to 14. The registration fee is $50. For details, write the Dean, Wayne University College of Medicine, 1512 St. Antoine Street, Detroit.

Prize for Ophthalmic Work.—The University of Buffalo awards annually a gold medal for a work on an ophthalmologic subject. Recent awards went to Dr. J. G. Bellows, of Chicago; Dr. Joseph Globus, of New York, and Dr. J. N. Evans, of Brooklyn. For details, write Dr. H. W. Cowper, 543 Franklin Street, Buffalo.

Obituaries

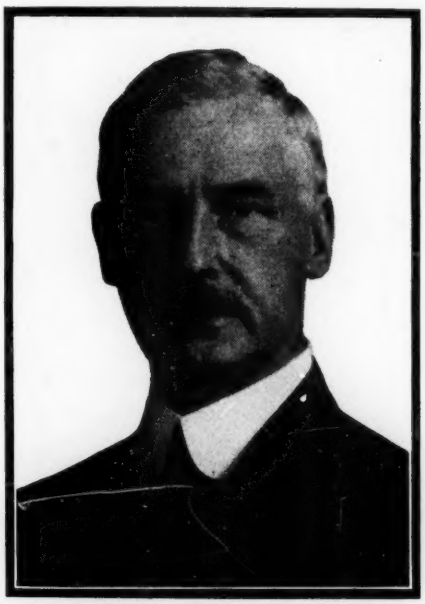
GEORGE EDMUND DE SCHWEINITZ, M.D.
1858-1938

Dr. George Edmund de Schweinitz died at his home in Philadelphia on Aug. 22, 1938, at the age of 79. Dr. de Schweinitz held the admiration and respect of all who knew him. He was tall and erect, with kindly blue eyes, and he possessed a voice that, while mild, had an agreeable and penetrating power that was unusual. His charm and powerful personality made him conspicuous in any group of men, and he well deserved the title so often given him—Dean of American Ophthalmology.

His ancestry goes back to Moravian nobility. He was born on Oct. 26, 1858, in Philadelphia, the son of Bishop Edmund de Schweinitz and Lydia de Schweinitz. He received the degree of Bachelor of Arts in 1876 and the degree of Master of Arts in 1878 from Moravian College, Bethlehem, Pa., and the degree of Doctor of Medicine from the University of Pennsylvania in 1881. He was first honor man in his class. After being graduated, he was resident physician at the Children's Hospital in 1881 and at the University Hospital from 1881 to 1883. From 1882 to 1887 he was quiz master on therapeutics at the Medical Institute, and from October 1883 until June 1888, prosector of anatomy at the University of Pennsylvania in the service of the late Professor Leidy. For a time he devoted himself to general practice, but soon, under the direction of the late Dr. William F. Norris, he began the study of ophthalmology. In 1885 he was elected ophthalmic surgeon to the Children's Hospital and in 1886, ophthalmologist to the Orthopedic Hospital and Infirmary for Nervous Diseases, where he was associated with the late Dr. Weir Mitchell. He was appointed ophthalmic surgeon to the Philadelphia General Hospital in 1887 and professor of ophthalology at the Polyclinic and College for Graduates in 1891. He resigned from the latter position in 1892, when he was elected clinical professor in ophthalmology at the Jefferson Medical College. In 1888 he was elected one of the editors of the *University Medical Magazine,* afterward published as the *University of Pennsylvania Medical Bulletin.* While editor of the *University Medical Magazine,* he served in this capacity first with the late Dr. Hobart A. Hare and later with the late Dr. Edward Martin. He was appointed professor of ophthalmology at the Jefferson Medical College in 1896, which position he held until 1902, when he was made professor of ophthalmology at the University of Pennsylvania.

In this position he served with distinction until 1924, when he resigned. He later became a member of the board of trustees of the university.

He was a member of the Pathological Society of Philadelphia, the Philadelphia County Medical Society, the Philadelphia Neurological Society, the College of Physicians, of which institution he was president from 1910 to 1913, the American Ophthalmological Society, of which he was president in 1916, and the American Medical Association, of which he was president in 1922. He was vice president of the Pennsylvania Institute for Instruction of the Blind at Overbrook and a member of the Academy of Natural Science, the American Philosophical Society, the Ophthalmological Society of the United Kingdom, the



GEORGE EDMUND DE SCHWEINITZ, M.D.
1858-1938

Société française d'ophtalmologie and the Société belge d'ophtalmologie; he was an honorary member of the Royal Society of Medicine of London and of the Hungarian and Egyptian ophthalmologic societies.

As one of the outstanding ophthalmologists in the world, Dr. de Schweinitz was honored at home and abroad. He was the recipient of the Alvarenga Prize in 1894, awarded by the College of Physicians of Philadelphia, for his essay on toxic amblyopia. He received the bronze plaque of the Société française d'ophtalmologie in 1923, the title of his address being "Ocular Manifestations of Focal Infections." The Howe Medal in Ophthalmology was awarded to him in 1927 and the Huguenot Cross in 1928. He gave the Bowman Lecture before the Ophthal-

mological Society of the United Kingdom in 1923, the title of his address being "Concerning Certain Ocular Aspects of Pituitary Body Disorders, Mainly Exclusive of the Usual Central and Peripheral Hemianopic Field Defects." He was awarded the Leslie Dana Medal for meritorious work among the blind, which was presented at St. Louis in 1930, when he delivered a notable address on "The Heritage of Sight; Its Conservation." The medal bore the inscription of "wise, learned, patriotic, teacher and guide." The University of Pennsylvania conferred on Dr. de Schweinitz the honorary degree of Doctor of Laws in 1914, the University of Michigan conferred the degree of Doctor of Science in 1922, and Harvard University conferred a similar degree in 1927.

Dr. de Schweinitz belonged to the Philadelphia Club, the University Club of Philadelphia and the University of Pennsylvania Club of New York.

His contributions to medicine were varied and epoch making. He was the author of many textbooks, brochures, treatises and scientific papers and did extensive research on the eye. He was the author of "The American Textbook on Diseases of the Eye, Ear, Nose and Throat," in collaboration with the late Dr. B. A. Randall, and of a standard work on "Toxic Amblyopias." His book entitled "Diseases of the Eye" went through ten editions and has been the outstanding American textbook for many years. At one time he was American editor of Haab's "Ophthalmoscopy and External Diseases of the Eye and Operative Ophthalmology." He wrote several communications in association with the late Dr. Thomas B. Holloway, one of the most important of which was on "Pulsating Exophthalmos," and with Dr. Edward Jackson he edited the "Ophthalmic Year Book" from 1905 until 1909.

When the United States entered the World War in 1917, Dr. de Schweinitz was made a member of the Council of National Defense and later entered the medical corps of the army as major and did meritorious work both in this country and abroad. After the war he joined the Medical Officers' Reserve Corps. and became a member of the editorial board of "The Medical Department of the United States Army in the World War." At the time of his death he held the rank of brigadier general in the Medical Officers' Reserve Corps.

He was president of the International Congress of Ophthalmology held in Washington in 1922, and at that time was designated as one of two to represent this country on an international committee, the object of which was to reorganize the International Congress of Ophthalmology. He took a prominent part in the deliberations of this committee and was later made a member of the council.

He enjoyed a large and influential private practice in addition to the usual opportunities for clinical and pathologic research in general and special hospitals with which he was connected. As a clinician, he was painstaking, thorough and skilful and never lost sight of the fact that

the eye and its diseases were closely allied to general diseases and diseases of the central nervous system. He displayed sound surgical judgment as an operator and did his work with meticulous care, always having the best interest of his patients at heart. He was an indefatigable worker and a diligent student and found little time for relaxation. He never married but devoted himself assiduously to his chosen profession. As a consultant, he held a most enviable reputation. His reports were meticulous, and his attitude toward the members of the medical profession was ideal. He was essentially the doctors' consultant. He was in great demand as a public speaker, and his after-dinner speeches were prepared with the same care that he gave his scientific addresses. He was fond of walking but never participated in any of the popular sports. His principal diversion was reading, and he liked a good detective story.

The Spartan type of character that was his was well illustrated in the manner in which he met his attack of glaucoma. Quite by accident he discovered that both his pupils were dilated. Investigation revealed that he had early simple glaucoma in both eyes. In spite of treatment the tension remained high, and he decided on an early operation. The trephine operation was performed on both eyes, and an excellent result was obtained. His answer to an inquiry concerning the effect of the disease on his vision was very descriptive. He said: "I can best describe my seeing ability by stating that where I previously took a glance I now have to take a look." His central vision remained nearly normal until the time of his death.

All who were privileged to know Dr. de Schweinitz had a real affection for him. His loss will be keenly felt by a large circle of acquaintances, while to his many friends his death leaves a void as teacher, author, clinician, surgeon, administrator and friend that will be impossible to fill. Sincere sympathy is extended to the remaining relatives and friends in their irreparable loss.

WALTER R. PARKER, M.D.

For the past fifty years George de Schweinitz was the most widely known and the most erudite of American ophthalmologists.

It is not necessary for me to catalogue his numerous services to his country or to list the many honors heaped on him by the societies and universities of his country and of foreign countries, because some of these are to be found in "Who's Who," but it may be of interest for me to recall an occurrence characteristic of the man, drawn from his and my intimacy, early established and steadily maintained for half a century. We had by appointment an annual meeting, either at his home, presided over by that remarkable woman his mother, or at his club. The purpose of the colloquy that followed was simply, *à la* Callimachus, "to tire the sun with talking and send him down the sky."

To me these informal trysts were inspiriting occasions. I feel that I owe to them much of whatever use I have been in the world; moreover, they permitted me to commune with the real de Schweinitz, who was not only a prince charming with a vivid personality but a Gamaliel at whose feet I sat in wonder and admiration. My friend that has gone was in the best sense of that term a great man whose memories "death, who taketh all away, *them* he cannot take."

CASEY WOOD, M.D.

Rome, Italy.

A mere catalogue of de Schweinitz's attainments gives an inadequate idea of his personality and of his outstanding position, not only in the history of ophthalmology but in the history of medicine in the United States. He was both a great surgeon and a great physician, and it was eminently apt that he should have held the presidency of the Philadelphia College of Physicians. His life was unremittingly devoted to ophthalmology and to the encouragement of medicine in the famous and old established University of Pennsylvania. An object which he had much at heart was the enrichment of the magnificent medical library in Philadelphia, and much of the time of his annual visits to England was devoted to seeking out medical incunabula in London and elsewhere. De Schweinitz belonged to the type of American one associates with New England but who also flourishes in Pennsylvania—that erudite and highly cultured type, whose distrust of humbug and anything cheap engenders a reserve which can be as impenetrable as armor plate. Only those who succeeded in overcoming this reserve can appreciate the sterling worth which it masked. To his accepted friends, de Schweinitz was a delightful companion and a staunch comrade.

As an Englishman who was proud of his friendship for many years, it is fitting that I should express the profound admiration all ophthalmologists on this side of the Atlantic—British and Continental—had for de Schweinitz. At the meetings of the Ophthalmological Society of the United Kingdom he was an honored guest, until in due time he became Bowman Lecturer and one of the few honorary members of the society. By his death the world has lost a great ophthalmologist and a great gentleman.

J. HERBERT PARSONS.

London, England.

As a student, de Schweinitz had been well prepared by heredity and teaching. His father, the Moravian bishop, had been able to give him a most excellent intellectual start, but he was unable to contribute largely to the financial side of his medical education. There was a legacy of

$500 left by his grandmother, but this was not sufficient; so to add to it he taught school for two years at Nazareth Hall, thus largely or perhaps altogether paying his own way. This, too, was good mental exercise, preparing him for his success as a teacher in later years.

While teaching school he became associated with a physician in Nazareth and got a good start in practical medicine. His popularity in his class at the university is shown by his election as class president. He made friends easily. He had no fads. In those days poker was a favorite indoor pastime. He was not at all fond of the game and was but an indifferent player. He was a good speaker and conversationalist, not given to florid oratory but employing careful, well chosen, convincing words. His writing was of the same nature, carefully thought out beforehand. He rarely had to alter a manuscript.

On his graduation his was the second class of the three year term. He was appointed intern at the Children's Hospital, largely through the friendship of Dr. John Ashhurst Jr. This internship ran for a year, but it was the usual custom to divide it into two terms. At the end of six months I followed him there, and he me to the University Hospital, this appointment being obtained as the result of a competitive examination.

After his year there he had a room with me in the house where I had started practice. He had not settled definitely what line to pursue, probably general practice, when an offer for an assistantship came from Dr. Norris. After studying over this offer for some time, he finally accepted it as a practical way of starting. Dr. Norris had watched him as intern and had recognized his capabilities.

De Schweinitz had no predilection toward ophthalmology; it was this offer that influenced him. Without it he would probably have drifted into surgery, for his preceptor, Dr. Wharton, was a surgeon, and he afterward held the position of surgical registrar at the University Hospital and was prosector of anatomy under Dr. Leidy. I think that his greatest objection to general practice was that he would have to do obstetric work. His first office was at 1330 Spruce Street, and here his luck held good in that among his fellow boarders were several men, slightly older than he, who had most excellent social contacts, and they, after recognizing his worth, introduced him to their friends, thus helping to start his social career which was such a success. At first he slept on the couch in his office, using the sitting room of the house as his waiting room. In the beginning he looked after all the general practice he could get in addition to his ophthalmic work. His income from practice was at first small. He added to it by sitting up at night with patients of older practitioners. This was before the days of trained nurses. He also established a successful quiz class on therapeutics among the students. To illustrate how close the wolf often was to his door, here is a story he used to tell.

He was walking home, since he did not wish to pay streetcar fare, when he met Dr. Agnew, who asked him to come to the hospital and administer ether to a patient. He received $10 for this and so was able to pay his office rent, about which he had been in some doubt. He had been in practice about six years before he could afford a secretary. He then employed one, at first part time and afterward constantly. She remained with him until his death and became quite a factor in the smooth running of his practice. All of his colleagues know her as Miss Mary. He changed his office from 1330 Spruce Street to 1401 Locust Street to establish a home for his stepmother and stepsister, his father having died.

Looking back over his life, one can see that his success was due to his native ability plus his unswerving devotion to his profession.

W. E. HUGHES, M.D.

LADISLAS DE BLASKOVICS
1869-1938

After receiving his medical degree in 1893 in Budapest, Hungary, Blaskovics began his ophthalmic training as assistant to de Grosz in the First University Eye Clinic, where he remained for ten years. He then acted as director of the Hungarian State Eye Hospital for twenty years. In 1928, on the death of von Hoor, he was appointed professor of ophthalmology and director of the Second University Eye Clinic in Budapest. In 1936, on the retirement of de Grosz, the two university clinics were combined, and Blaskovics acted as director, though his health was far from good. He died on Oct. 17, 1938, of pneumonia.

His talents lay in the surgical field. It is stated that even in his student days his dexterity in operations on animals was remarkable. The wealth of material in the University Hospital in Budapest gave him the opportunity of developing many new operative procedures and established his reputation as one of the great ophthalmic operators in the world. He was particularly interested in plastic surgery, in which field he was a pioneer and devised many new operative procedures. He is perhaps best known for his operation for ptosis. Although he was first opposed to the intracapsular operation for cataract, he later became an enthusiastic convert and suggested a special capsule forceps for this procedure. It was a revelation and a pleasure to watch him at work in the operating room. Always quiet and cool, working with practically no assistance and without fuss and helping himself to the instruments which were spread out on a side table, he conducted his operations on the soundest surgical principles and without trauma. He also made contributions to other fields of ophthalmology and devised a new unit for measuring vision, oxyoptry, in which a 1 degree angle replaced that of 1 minute. He wrote a text on operations on the eyes in Hungarian, which appeared in several editions. An enlarged edition of this book in German, written in conjunction with A. Kreiker, appeared only a few months ago.

ARNOLD KNAPP.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Biochemistry

CHEMICAL AND HISTOCHEMICAL INVESTIGATION OF UREA IN THE LENS AND OCULAR MEMBRANES. I. BORSOTTI, Ann. di ottal. e clin. ocul. **66:** 32 (Jan.) 1938.

The xanthydrol test of Fosse as modified by Vercellana was employed, crystals of xanthic urea being formed in the section of the tissue under investigation. When the whole eye of the rabbit was fixed with xanthydrol, crystals were found in the vitreous, cornea, sclera and retina, especially in the internal layers. When the cornea alone was removed before the death of the animal, however, and fixed in the same way, no urea crystals were found. This indicated that the crystals found in the cornea of eyes fixed after death are due to rapid post-mortem diffusion from the aqueous, which does not take place during life while the cornea is exercising its function of selective permeability. The same is apparently true of crystals found in the sclera and retina, since when the aqueous and vitreous were rapidly removed before death, or when the fixative was injected into the globe during life few crystals were found in the sclera or retina.

These observations suggested that the findings of Schmorl on the urea content of the crystalline lens might be in part explained by post-mortem diffusion. To control this factor, the author estimated urea quantitatively in the lenses of cattle, sheep and rabbits extracted immediately after enucleation of the eye and twelve hours after enucleation. The lenses extracted immediately showed a urea content of 16.1, 18.1 and 13.9 mg. per hundred cubic centimeters for beef, sheep and rabbit lenses, respectively. When left in the eye twelve hours before extraction of the lens, the figures were 22.9, 20.4 and 21.8 mg. per hundred cubic centimeters. The definite increase in urea in the lens must be attributed to increased permeability of the lens capsule occurring after death. The author believes that his work offers additional proof of the selective permeability of certain living tissues, such as the cornea and lens capsule.

S. R. GIFFORD.

Congenital Anomalies

CONGENITAL ANOPHTHALMOS VEILING AN OPHTHALMOTERATOID: REPORT OF A CASE. E. SCHRECK, Klin. Monatsbl. f. Augenh. **100:** 74 (Jan.) 1938.

Schreck reports in detail the clinical, operative and histologic data with reference to a girl aged 17 who was examined at the ophthalmic clinic of Heidelberg University for absence of her left eye at the age of 10 days. At that time congenital anophthalmos with a vicarious orbital tumor was found. A plastic operation, performed when the girl was 17 years old, revealed a tumor, the size of a walnut, which was con-

nected with a coloboma of the lower lid. The tumor presented the pathologic picture of an early malformation of the ocular "anlage" and a teratoid formation.

The relation between congenital anophthalmos and teratoma is discussed and evaluated; the result of this evaluation was a separation of congenital anophthalmos into genuine and apparent. The diagnosis of the genuine type should be made only after exact microscopic examination of the orbital contents reveals the total absence of ocular rudiments. All other cases should be recorded under the group of apparent anophthalmos, which type forms a transition to microphthalmos, in which remnants of the globe may be recognized microscopically.

In Schreck's case there was complete absence of ocular rudiments, but pathologicoanatomic proof was furnished of the presence of an undeveloped and malformed anlage of the sclera, choroid and retina. Glia cells were found in abundance, and their provenience was doubtlessly traced to retrograding retinal tissue. In addition, a teratoid tumor was found in the orbit which consisted of underdeveloped derivates of two embryonic layers. This tumor did not belong to the fourth group of teratomas of Mizno, as it did not contain any tissues which might not occur in a malformed ocular anlage. Other considerations, too, pointed to a tumor derived from an ocular malformation. Schreck thinks that in his case an early disturbance of the ocular anlage produced a severe ocular malformation, associated with a teratoid tumor in the orbit, which belongs to the group of the apparent anophthalmos.

K. L. STOLL.

Cornea and Sclera

THE TREATMENT OF INFECTED ABRASIONS OF THE CORNEA. C. F. KUTSCHER, Pennsylvania M. J. **41**: 979 (Aug.) 1938.

Kutscher calls attention to the value of sodium hypochlorite in the management of infected abrasions of the cornea. The chemical is employed in a strength of 1: 5,000 and is instilled every second hour day and night for twenty-four hours. After twenty-four doses, the instillations are made every fourth or sixth hour. If the infection is not checked, delimiting keratotomy is done.

W. ZENTMAYER.

KRUKENBERG'S PIGMENTED SPINDLE AND THE PIGMENT RING IN THE CORNEA: REPORT OF CASES. SOMMER, Klin. Monatsbl. f. Augenh. **99**: 468 (Oct.) 1937.

Sommer reports 3 cases of disseminated pigmentation on the endothelium of the cornea associated with other symptoms. The first patient, a woman aged 31, presented bilateral Krukenberg spindles in the presence of an otherwise perfectly normal cornea and normal vision. The second patient, a woman aged 61, had similar bilateral spindles, which, however, were less distinct than those of the first patient. No changes were observed after the extraction of cataracts from the eyes, which were myopic. The patient did not have diabetes. In the third case the spindles were associated with chronic glaucoma. They became less conspicuous after bilateral trephining after the manner of Elliot. The atrophic retina in these slightly hyperopic eyes showed numerous scattered pigment cells. Tension was normal after the operations, but

the cupping of the disks persisted. The cause of the spindles is discussed by the author, who arrives at no definite conclusions.

K. L. Stoll.

Experimental Pathology

An Experimental Study of Choked Disc in the Rat. W. A. Jeffers, J. Q. Griffith Jr., W. E. Fry and A. G. Fewell, Am. J. Ophth. **20:** 881 (Sept.) 1937.

On the basis of experiments on rats, the authors give the following summary:

"1. Rats developing increased intracranial pressure and vascular hypertension following the intracisternal injection of kaolin were found to develop no ocular changes upon ophthalmoscopic or histologic examination.

"2. Ten rats developed a condition simulating papilledema upon the implantation of cerebellar tumors, as confirmed by the same examinations.

"3. Eight rats that were known to have increased intracranial pressure after the intracisternal injection of kaolin failed to develop signs of papilledema in the presence of large cerebellar tumors.

"4. The evidence presented is interpreted as support for the view that papilledema is due to an increased flow of fluid from the cerebrospinal space to the eye, and that it is not due to a block in the flow from the eye toward the cerebrospinal space.

"5. Our findings are discussed with reference to hypotheses concerning the nature of papilledema."

W. S. Reese.

Histologic Study in Experimental Wounds and Sutures of the Optic Nerve. C. Tita, Ann. di ottal. e clin. ocul. **66:** 51 (Jan.) 1938.

The author sectioned one optic nerve of each of 60 rabbits with care to avoid the central vessels. The fragments were held in close apposition by a suture and by sparing the posterior portions of the sheath. Sections were made at various intervals from three to thirty days later. Regeneration of fibers could be observed by the seventh day and was more extensive by the fourteenth and twenty-first days. By the twenty-first day, a few fibers could be traced through the cicatricial zone. By the thirtieth day, however, secondary degeneration was already evident in the regenerated fibers, which were obviously being compressed by condensation of the scar tissue. Studies of the later processes have been made by Rossi, who showed further degeneration of the new fibers and never saw fibers traverse the scar. The author concludes that regeneration of fibers in a functional sense after grafting of the eye is entirely impossible.

S. R. Gifford.

General

Anniversary Number. Ann. d'ocul. **175:** 1 (Jan.) 1938.

In January 1938 the *Annales d'oculistique* reached the hundredth anniversary of its foundation. In January 1838 Florent Cunier issued

the first number, and in the present issue is found a complete table of the articles published in the first volume with an exhibit of the title page. There are also photographs of Cunier, Warlomont, Desmarres, Dor, Panas, DeWecker, Gayet, Javal, Badal, Abadie, Coppez, Nuel, Parinaud, Landolt, Lagrange, Truc and Van Duyse. There are also articles on "A Hundred Years of Franco-Belgian Ophthalmology" by Rochon-Duvigneaud; "Paralysis of Associated Movements of the Eyes," published by Parinaud in 1883; "Iridectomy and Sclerectomy Combined in the Treatment of Chronic Glaucoma: New Procedure for the Establishment of a Filtering Cicatrix," published by Lagrange in 1906; "Vision" by Nuel, published in 1904; and an unpublished article by Morax on "Gonococcus Infection."

The editorial board of the present *Annales* consists of Magitot, chief editor; Rochon-Duvigneaud, Bailliart, Hartmann, Leplat, Dupuy-Dutemps, Redslob, Jeandelize and Hambresin.

S. H. McKee.

General Diseases

Tularemia from the Clinical, and Especially from the Ophthalmologic, Standpoint. A. Jess, Klin. Monatsbl. f. Augenh. **99**: 577 (Nov.) 1937.

The advance of tularemia in Germany prompted Jess to report on the symptoms of this disease in a short, but precise article. The oculist is first consulted in many cases, as the ocular symptoms frequently prevail. Trachoma and tuberculous conjunctivitis will have to be considered from the point of view of the differential diagnosis. The early general symptoms, such as fever, malaise, lack of appetite and involvement of the preauricular and submandibular glands, are characteristic. The glands may remain swollen for weeks after the ulcers of the conjunctiva disappear. This oculoglandular form resembles Parinaud's conjunctivitis, the origin of which has been traced to animal infection, as has been tularemia. Three other forms of tularemia occur: the ulceroglandular form, which begins with ulcers in the epidermis and swelling of the glands; the glandular form, in which infection of the glands presents first, and the typhoid, or the septic form. Conjunctivitis infectiosa necroticans, described by Pascheff, appears to be related to tularemia. The death rate from tularemia is 4 per cent according to Jess. The port of entrance is the eye, and in many cases the disease is apparently due to eating insufficiently cooked meat of infected animals. Cases of tularemia have been reported in the United States, Sweden, Norway, Japan and Russia and recently in the Balkans, Moravia, Austria and Bavaria. A total of 396 cases of human tularemia were recorded in Bohemia up to the end of March 1937. Aside from rabbits and other rodents, pheasants, partridges, foxes, dogs, cats, hogs and sheep are subject to tularemia. Human beings may become infected by furs. The exact anamnesis, inoculation of animals, an agglutination test and a test for cutaneous allergy are essential in arriving at a correct diagnosis. The agglutination test is not positive prior to the second week. Infection from man to man has not been observed. The therapy consists of the use of antiserum and local ocular treatment. Permanent damage to the eyes has not been reported.

K. L. Stoll.

SPECIFIC DIAGNOSIS OF TUBERCULOUS DISEASES OF THE EYES: REPORT OF CASES. W. ROHRSCHNEIDER, Klin. Monatsbl. f. Augenh. **99:** 682 (Nov.) 1937.

In cases of miliary and other forms of tuberculosis of the uvea the diagnosis of ocular tuberculosis may be readily arrived at by the observation of the clinical picture. In many ocular diseases, however, which are ascribed to tuberculosis, diagnostic tests, such as roentgenograms of the lungs, serologic reactions and intracutaneous and subcutaneous tests, are not reliable. The evaluation of focal reactions may be still more difficult. This evaluation seems to be facilitated by observation of the intraocular pressure, which drops after injections of tuberculin used for diagnostic purposes. Rohrschneider refers to the research in point done by Samojloff and Mexina, who, he contends, have not considered spontaneous undulations of the intraocular pressure in inflamed eyes. Such undulations occurred in the third case described. Rohrschneider shows tables with data on the intraocular tension and temperature for 20 patients. He concludes that undulations of the intraocular tension after injections of tuberculin cannot be depended on for the diagnosis of a tuberculous focal reaction. It is necessary, in his opinion, to observe the intraocular tension for a few days prior to the diagnostic injection of tuberculin. The tension barely changed or did not change at all in some cases presenting clinical evidence of an ocular focal reaction of tuberculosis; his second case furnishes proof of this observation. Therefore, undulations of the intraocular tension after tuberculin tests are of limited use in the diagnosis of tuberculosis.

K. L. STOLL.

Glaucoma

THE PHOTOCHROMATIC INTERVAL IN GLAUCOMA AND CAVERNOUS ATROPHY. R. PICKARD, Brit. J. Ophth. **22:** 391 (July) 1938.

The author undertook the examination of the photochromatic interval in cases of glaucoma and cavernous atrophy. The photochromatic interval is the condition in which a color produces stimulus of light but not of color. The test was applied by moving the stimulus from the periphery of the field to fixation. The author concludes that it may fairly be said that if the red field is diminished and the photochromatic interval is under 5 degrees, there is a five to one chance that the condition is glaucoma. Conversely, if the red is reduced and the photochromatic interval is over 5 degrees, the chances are one to four that it is cavernous atrophy.

The article is somewhat technical and should be read in the original.

W. ZENTMAYER.

CONTROL OF THE INTRAOCULAR PRESSURE IN INFANTILE GLAUCOMA. W. KAPUSCIŃSKI, Arch. f. Ophth. **138:** 673 (May) 1938.

The object of the studies on which this paper is based was to determine in which forms or in what phases of glaucoma atropine is not contraindicated or even indicated. First, the author confirmed the well

known fact that mydriatics or miotics exert no appreciable influence on the intraocular tension of normal eyes of children. In 3 cases of typical hydrophthalmos (children from 2 to 6 years of age) the tension with the use of atropine was certainly no higher than that when no medication was used or when miotics were employed. In 1 case of hydrophthalmos combined with nevus flammeus of the face pilocarpine had practically no effect, whereas the first dose of atropine after a period during which miotics were used several times produced an acute though transient drop in pressure. Still more striking was the pressure-lowering effect of atropine in a case of glaucoma simplex (a girl aged 20) in which cyclodialysis of each eye was necessary five times in order to bring about lasting hypotony (tension of each eye, between 8 and 13 mm. of mercury). About a year after the last cyclodialysis, an acute attack of glaucoma suddenly developed in one eye with a tension of 58 mm. of mercury and a deep anterior chamber. Pilocarpine narrowed the pupil without any effect on the tension. After one dose of atropine the pupil dilated and the tension dropped at first to 26 and, a few hours later, to 6 mm. of mercury. During a two months' stay in the hospital, the patient had six more acute attacks, each of which was promptly controlled by the use of atropine. A striking subjective symptom which preceded each attack was a sudden lowering of the refraction caused by deepening of the anterior chamber. P. C. KRONFELD.

Hygiene, Sociology, Education and History

LIGHT RESERVE FOR OCCUPATIONS IN SIGHT-SAVING CLASSES. R. A. KAZ, Brit. J. Ophth. **22:** 482 (Aug.) 1938.

Kaz emphasizes the role of illumination in relieving the ocular and reflex symptoms of ocular fatigue. W. ZENTMAYER.

CHILDREN WITH DEFECTIVE VISION, IN NEED OR NOT OF SIGHT-SAVING CLASS. R. A. KAZ, Brit. J. Ophth. **22:** 486 (Aug.) 1938.

Kaz points out that treatment directed to the cause of the reduced vision which has resulted in a child being relegated to a sight-saving class may in time bring sufficient improvement to permit the child to return to schools for normal sighted pupils. W. ZENTMAYER.

INSTRUCTION AND PROFESSIONAL ORIENTATION OF AMBLYOPIC CHILDREN: CREATION OF SPECIAL SCHOOLS. V. ORTIZ, Arch. de oftal. de Buenos Aires **13: 7** (Feb.) 1938.

In this article an appeal is made for the establishment of special schools for the education of amblyopic children. Such schools were first established in Great Britain by Bishop Harman and James Kerr. The author specifies the conditions required for admission and for the selection of the teaching personnel and the advantages to be derived therefrom.

The teaching methods are next detailed, with classification of the pupils into elementary, medium and high groups. A good general education and proper manual training are advocated, with stress on the importance of the proper selection of a profession. C. E. FINLAY.

Injuries

SURGICAL TREATMENT OF CAUSTIC BURNS OF THE EYE: REPORT OF CASES, WITH ILLUSTRATIONS. O. THIES, Klin. Monatsbl. f. Augenh **99:** 764 (Dec.) 1937.

In an exhaustive manner Thies describes the caustic injuries of the face, lids and eyeball. A detailed synopsis is given of the evolution of the surgical treatment which was begun by Thiersch and was continued and improved by such men as Kuhnt, Czermack, Stellwag, Axenfeld and others. Short histories are added of a number of cases observed by Thies, which are illustrated by 21 pictures. The course of caustic injuries is described, and the opinion is expressed that only surgical procedures at an early stage will produce results. Early transplantation of the mucous lining of the lip is considered essential. It is evident by the description rendered that all the authors had to follow their own road in the quest for a useful surgical method, many of which were replaced by more practical ones later. The plastic operations on the lids have been perfected to such an extent today that Thies can claim 100 per cent successful results. The duration of extensive plastic operations has been reduced from two hours to one-half hour, with the assistance of one nurse. Several cases are reported of caustic burns of the eyeball. Early plastic operation after Denig's method succeeded in removing disfiguring scars, in saving the sight of some patients and in stopping the progress of destruction in the cornea. The number of these injuries has been greatly reduced in recent years by forced precautionary measures at the factories.

K. L. STOLL.

Lacrimal Apparatus

SOME RESULTS OF INTRANASAL DACRYOCYSTORHINOSTOMY. T. E. WALSH and L. BOTHMAN, Am. J. Ophth. **20:** 939 (Sept.) 1937.

Walsh and Bothman give the following summary and conclusions of their experiences with intranasal dacryocystorhinostomy:

"Twenty-one tear sacs have been operated on by intranasal dacryocystorhinostomy with 85.7 per cent successful results.

"For cases of dacryocystitis with obstruction at the lower end of the sac, operation via the nasal route is preferable to dacryocystectomy, which does not often relieve tearing, and to the Toti operation, which leaves a scar."

W. S. REESE.

SYPHILIS OF THE LACRIMAL SAC SIMULATING ACUTE DACRYOCYSTITIS. P. DESVIGNES, Bull. Soc. d'opht. de Paris **50:** 13 (Jan.) 1938.

Syphilitic lesions in the vicinity of the lacrimal sac are rare, the chancre being exceptional. Periostitis with exostosis occurs at times during the tertiary stage. Cases of gummatous tumor of the lacrimal sac were reported by Kalt. Rollet and Colrat reported 1 case and Michaesco reported 3. The latter are similar to Desvignes' case in that the mass was soft and painful. When first seen, the patient, 36 years of age, complained that the condition had been present for a

number of days. The lesion had all the characteristics of acute dacryocystitis. Hot applications had little effect, and on incision much blood and a few drops of yellowish, viscid fluid gushed forth. The red tumefaction, a slight amount of pain and a fistula persisted. On exploration of the fistula, a gummatous process in the ethmoid bone was noted. The necessity of a nasal examination and the elimination of a possible diagnosis of tuberculosis are emphasized.

L. L. MAYER.

Lens

TOTAL EXTRACTION OF THE OPACIFIED CRYSTALLINE LENS WITH SNELLEN'S LOOP. F. P. ALLENDE, Arch. de oftal. de Buenos Aires **13:** 89 (Feb.) 1938.

After reference to the current methods of extraction of the lens in toto, the author describes his method of extraction with Snellen's loop. He reports 15 cases in which good results were obtained.

C. E. FINLAY.

EXPERIMENTAL STUDIES ON THE CONTRACTILITY OF THE LENS FIBERS. P. GILLESSEN, Arch. f. Ophth. **138:** 598 (May) 1938.

The anatomists Leuwenhoek and Pemberton called the lens "musculus crystallinus" because they thought that the lens fibers were contractile. At the beginning of this century this view was revived by W. Krauss; his pupil Gillessen now reports experiments in which small quantities of acetylcholine or histamine (80 micrograms of active substance, not stated in how much water) were injected into the equatorial portions of the lenses of rabbits, cats, dogs and monkeys. The animals were under a general anesthetic and had received a certain amount of atropine (obviously not enough to produce cycloplegia). The intralental injections were made with a glass capillary, which was introduced through the periphery of the cornea into the anterior chamber and from there through the dilated pupil in the periphery of the lens. If the pupil remained the same width during the injection and for a half hour after it, the author concluded that all of the active substance had been deposited in the lens and that none of it had escaped into the aqueous. The refraction was determined skiascopically before and several times after the injections. Fifteen minutes after the injection the author found a considerable increase, and half an hour later a decrease, in the refraction of the eye treated in this way. "Acetylcholine and histamine, if deposited in the lens cortex near the equator, are capable of eliciting contraction and relaxation of the lens fibers. The lens fibers are, therefore, no plasmatic tubes nor elastic fibers, but contractile elements similar to smooth muscle fibers."

P. C. KRONFELD.

Methods of Examination

BINOCULAR FIXATION FOR SCOTOMETRY AT ONE METER. J. FOSTER, Brit. J. Ophth. **22:** 426 (July) 1938.

Binocular fixation at longer distances than 33 cm. is still rather a problem, and existing apparatus is likely to interfere with the field of the examined eye, to be somewhat inaccurate or to be complicated

and expensive. The author states, however, that Cowan and Markov have produced a simple method which overcomes these difficulties, but their device is somewhat unwieldy. Pinned to the Bjerrum screen is a gray circle 5 inches (12.7 cm.) in external diameter, painted on a 7 inch (17.8 cm.) square of black paper, so that the center of the circle overlies and conceals the fixation point on the screen. The screen should have an even illumination of at least 7 foot candles. The second part of the apparatus consists of the cross projector, resembling an ordinary ophthalmoscope. The patient holds the instrument in one hand and sees an illuminated image of a cross which appears as if it were situated on the Bjerrum screen. The article is illustrated.

W. ZENTMAYER.

Neurology

HYPERTENSIVE MENINGEAL HYDROPS. L. M. DAVIDOFF and C. G. DYKE, Am. J. Ophth. **20**: 908 (Sept.) 1937.

A syndrome characterized by increased spinal fluid pressure, headache and papilledema is discussed, the literature is reviewed and 15 cases are presented. The authors suggest that this syndrome be called "hypertensive meningeal hydrops." The characteristics to be noted in the pneumoencephalogram are: a normal ventricular system, difficulty in filling the ventricles during ventriculography and the escape of gas into the cisterna subarachnoidales and cerebral sulci during ventriculography. Subtemporal decompression is an important therapeutic measure, especially if elevation of the optic disks is great and the patient's vision is threatened. The prognosis is uniformly good, although the cure does not occur rapidly. The authors believe that it takes months or even years before a normal equilibrium between production and absorption of the cerebrospinal fluid is established.

W. S. REESE.

OPTOCHIASMIC ARACHNOIDITIS DUE TO PNEUMOCOCCUS. M. GUILLERMIN and J. PESME, Bull. Soc. d'opht. de Paris **50**: 16 (Jan.) 1938.

A young soldier in the course of his training noticed a sudden and marked loss of vision. Examination showed bilateral choked disks, greater on the right side, an absence of hemorrhages and wide but reactive pupils. The visual acuity in the right eye was 1/100 and in the left eye 1/50. The results of the entire general examination were negative. Exploratory visualization of the optic chiasm revealed marked thickening and redness of the arachnoid. When the adhesions were broken, the cerebrospinal fluid in this region appeared cloudy. Recovery was complicated by epileptiform seizures and an increase in temperature. Frequent spinal taps showed cloudy fluid, which, when cultured, revealed a strain of pneumococcus. The patient died, and on the basis of the results of autopsy the authors are of the opinion that a localized pneumococcic arachnoiditis was disseminated by operative intervention.

L. L. MAYER.

Ocular Muscles

ON SO-CALLED REFUSION. K. RAAB, Klin. Monatsbl. f. Augenh. **99:** 475 (Oct.) 1937.

Haploscopic research showed that accommodation and convergence fail often in adjusting the visual axes for the intended point of fixation, even in the presence of emmetropia and orthophoria. This so-called physiologic exophoria is compensated by fusional convergence. The convergence may be increased beyond the necessary measure, while the degree of accommodation remains unaltered. The rotations possible in obtaining increased or decreased convergence, while the accommodation remains unchanged, is called the relative width of convergence. The upper limit of relative convergence used to be considered the point at which the convergence changed into diplopia. Pascal found that prior to the advent of diplopia a moment arises in which the binocularly observed single picture appears indistinct. This indistinctness is due to excess accommodation and may be overcome by means of weak concave lenses. The moment when the indistinctness appears is the actual upper limit of the relative convergence. The indistinctness disappears, and two clear pictures will be observed if fusion is prevented by forcing exaggerated convergence. The double images will fuse as soon as the prisms are reduced. This moment is called the refusion point, which is identical with what Pascal called the recovery point. The results of the research done by Pflimlin, Strübin and others are discussed.

Raab arrived at the following conclusions: The refusion point depends on the existing tonus of the muscle, which presents individual differences. The position of the point of indistinctness, which is the upper boundary line of the width of relative convergence, determines the amount to which the tonus may be increased. A primary and a secondary point of refusion can be distinguished in some persons. The position of the primary point can be determined by a persistence of an increased accommodation on convergence, while the secondary point depends on the tonus of the muscle. There are simple hypotonic forms and accommodatively compensated hypotonic forms of insufficiency of convergence; these differ clinically. The crossing of the curves of indistinctness and refusion, as described by Pflimlin and Strübin, are not the rule.

K. L. STOLL.

Physiology

ANISEIKONIA IN EMMETROPIA. W. L. HUGHES, Am. J. Ophth. **20:** 887 (Sept.) 1937.

This is a study of selected patients examined for aniseikonia at the New York Eye and Ear Infirmary. Hughes arrives at the following conclusion:

"The relative size of the ocular images of a group of 43 patients having symptoms that were thought to be ocular in origin was determined. In 14 the images were equal. In 29 they were asymmetrical (aniseikonia).

"The results in 14 cases in which complete data were obtainable are reported. Relief was obtained in 10 by the wearing of iseikonic zero power lenses designed to correct the aniseikonia present. It has been shown that the presence of aniseikonia is independent of any refractive error."

W. S. REESE.

Retina and Optic Nerve

ANATOMICOPATHOLOGIC RESEARCHES ON A GLOBE WITH PARTIAL RETINAL DETACHMENT OF TWENTY DAYS' DURATION. MAGITOT and LENOIR, Bull. Soc. d'opht. de Paris **50:** 34 (Jan.) 1938.

The eye of a young woman 35 years of age was removed because an intraocular tumor was suspected. Loss of vision was noted suddenly without a history of trauma. The media were clear, the tension was normal, and the detachment was located temporally near the disk. No holes or hemorrhages were found, but a few white spots on the surface and a slight haziness on transillumination were enough to cause tumor to be suspected.

Cystic degeneration was noted in the macular area. The retinal rods and cones were in good condition. The pigment epithelium showed some curious lesions. Islands of atrophy were found in the detached area together with degeneration of the hyaloid fossa. In places, the pigment epithelium seemed to have entirely disappeared. The choroid was in good condition. The suprachoroidal area was filled with an albuminous fluid. The authors have no hypothesis as to the causative agent in idiopathic retinal detachment but simply wish to offer their observations for what they are worth. L. L. MAYER.

RETINAL BLOOD PRESSURE IN CASES OF NASAL DISEASES AND THE EFFECTS OF SURGICAL INTERVENTION IN THE NOSE AND NASOPHARYNX. CATTANEO and LASAGNA, Ann. di ottal. e clin. ocul. **66:** 1 (Jan.) 1938.

Pressure in the retinal arteries was recorded with Bailliart's dynamometer and in the capillaries by entoscopy during pressure with the same instrument. Although actual measurements are not given, a more or less marked hypertension in the retinal vessels as compared with the brachial blood pressure was noted in 57 per cent of persons with various nasal diseases. Immediately after nasal operations, the incidence of relative hypertension rose to 76 per cent, while after a month or more only 27 per cent of the patients were found to show relative hypertension. This is considered to illustrate a vasomotor reflex between the retinal vessels and the nasal mucosa. S. R. GIFFORD.

DISCIFORM MACULAR DEGENERATION OF JUNIUS-KUHNT. G. FAVALORO, Ann. di ottal. e clin. ocul. **66:** 81 (Feb.) 1938.

The author reports 3 cases of disciform macular degeneration in patients 60, 53 and 55 years of age. The condition in 1 case was seen in an early stage, with little elevation of the newly formed tissue. That in the other cases was typical of a more advanced stage. In 1 case only one eye was affected. All patients showed a moderate degree of hypertension. The literature is reviewed, and especial attention is given to the few cases in which histologic examination was made. On account of its characteristic ophthalmoscopic picture, the author believes that the condition is a definite clinical entity which should be distinguished from other changes affecting the macula. He considers senile macular disease and familial macular disease to be a purely degenerative process,

while the exudative processes described by Axenfeld as retinitis externa is of an inflammatory nature. Disciform macular degeneration, he believes, shows a primary reactive transudative phase, followed by degenerative changes. The part played by the choroid in the early stages is emphasized.

S. R. GIFFORD.

MEASUREMENTS OF THE CALIBER OF THE RETINAL VESSELS OF PATIENTS WITH HYPERTENSION AND NEPHRITIS. G. BADTKE, Klin. Monatsbl. f. Augenh. **99:** 655 (Nov.) 1937.

In this detailed paper Badtke renders an account of the technical considerations of measurements of the retinal vessels. He adds a short and clear division of the types of hypertension and nephritides and of their symptoms. Tables are presented which show notes on the condition of the disk, the retina and its vessels and the blood pressure. The caliber of the retinal vessels was measured with Lobeck's *Messokular*. Table 5 shows the width of the retinal veins and the relation of the veins to the arteries in the various types of hypertension and nephritis. The results of Badtke's research follow: The width of the retinal arteries was smaller than that of the veins of healthy persons, even if the difference was slight. The arteries were somewhat wider than normal in cases of red hypertension. In some cases the artery was wider than the vein. The arteries were never contracted in cases of genuine red hypertension. Transition from benign red hypertension to malignant pale hypertension was evidenced by contraction of the arterial system. It was evident in the periphery in the beginning but could not be measured with Lobeck's method. The arteries near the disk showed unevenness of the caliber and occasional contraction for certain distances; these could usually be measured well with Lobeck's method. The contraction of the arteries was most evident in cases of complete pale hypertension. In cases of chronic nephritis no contraction of the arterial system was noted as long as the systolic and diastolic pressure remained low and as long as no ophthalmoscopic changes were present; the measurements equaled those found in healthy persons. The picture resembled that of red hypertension in some cases of high systolic blood pressure. Measurements of the caliber in these cases showed slight distention of the arteries.

K. L. STOLL.

CONGESTION OF THE RETINA INDICATING INVOLVEMENT OF THE RETINAL VESSELS IN FAILURE OF THE RIGHT SIDE OF THE HEART: REPORT OF CASES. G. JANKE, Klin. Monatsbl. f. Augenh. **99:** 756 (Dec.) 1937.

Janke refers to a previous report of a case of cyanosis of the retina which was not coupled with polycythemia (*Klin. Monatsbl. f. Augenh.* **96:** 605 [May] 1936). He reports 3 similar cases of congestion of the retina in which polycythemia, hitherto considered a regularly concomitant symptom of this disease, was absent. The patients suffered from emphysema of the lungs and cardiac failure. The circulation of their pulmonary blood was retarded by atrophy of the alveolar walls, lack of capillaries and inactivity of the thorax. This condition produced

compensatory hypertrophy of the right side of the heart, followed by dilatation of the chamber on that side. The result was retention of an amount of residual blood during each systole, as the heart was unable to handle the normal quantity of blood. As a consequence, stasis developed, which finally extended from the right atrium of the heart to the veins of the body. Simultaneously with asphyxia, cyanosis and congestion of the liver, considerable increase of the pressure in the venous system was noted. Primary polycythemia existed in none of the 3 cases, and secondary polycythemia of a low degree in 1. A high degree of distention and tortuousness of the retinal veins was observed in each case. The disks were slightly congested in the cases of milder involvement, and edema and hemorrhages of the disks and retina were present in the cases of severe involvement. The gravity of the pathologic changes in the fundus permit no prognostic conclusions. Decompression of the vascular system followed treatment in juvenile patients. Janke disagrees with Sobański, who believes that choked disk will result from lack of correlation between the increased venous tension due to increased cerebral tension and the arterial pressure in the retina. It is Janke's opinion that in congestion of the retina the venous stasis is primary and the edema of the disk secondary; in choked disk, on the other hand, the edema of the optic nerve is primary and the venous stasis secondary.

K. L. Stoll.

Treatment of Retinal Detachment by Means of Diathermy and Injection of Air into the Vitreous. B. Rosengren, Acta ophth. **16:** 3, 1938.

Since 1930 operations for the relief of retinal detachment have been performed with increasing frequency. In spite of improvements in technic, there has not been a corresponding increase in the number of cures obtained. They amount to about 50 per cent, as in Gonin's time. The author has interested himself in the reason for the numerous failures which still occur. They may be attributed, he believes, not to improper localization of retinal tears or inadequate cauterization but to failure to secure proper apposition of the retina to the underlying structures. Therefore, in addition to the usual procedures of diathermy and drainage, the author injects 1 or 2 cc. of air into the vitreous. The bubble acts as a tampon, which exerts pressure on the retina and serves to prevent seepage of vitreous through the retinal tear. The air itself, because of factors of capillarity, does not penetrate beneath the retina. It is of the utmost importance to keep the air bubble in contact with the retinal tear. This is effected by means of appropriate sutures beneath the rectus muscles and by suitably altering the position of the patient. Arruga's failures with injection of air are attributed by the author to nonobservance of this detail. The air is injected by means of a needle 0.36 mm. in diameter, inserted 5 or 6 mm. behind the limbus.

In a series of 20 operations on 18 patients cures were obtained in 14 instances.

O. P. Perkins.

Trachoma

STUDIES ON THE INFECTIVITY OF TRACHOMA: VII. FURTHER OBSERVATIONS ON FILTERABILITY OF THE INFECTIOUS AGENT. L. A. JULIANELLE, M. C. MORRIS and R. W. HARRISON, Am. J. Ophth. 20: 890 (Sept.) 1937.

This is the seventh article on the filtrability of the infectious agent of trachoma. The following summary and conclusions are given:

"1. Further studies have been made on the filterability of the infectious agent of trachoma, employing Berkefeld (kieselguhr), Elford (collodion), Kramer (plaster of Paris), and Seitz (asbestos) filters.

"2. In all of five experiments on filtration of infectious tissues through Elford filters alone, the filtrates were noninfectious.

"3. In seven additional experiments with active material, parallel filtrations with all four filters yielded on two occasions filtrates which were infectious, both resulting from Berkefeld V filtrates only.

"4. Filterability has been demonstrated, therefore, in two of 12 trials, as compared with one of 11 trials in a former study.

"5. The evidence indicates that the infectious agent of trachoma is irregularly filterable, and that the difficulties in filtration may be attributable to variations in the infectious tissues themselves, as well as in the composition of the filter employed.

"6. The possible factors involved in unsuccessful filtration are discussed and a hypothetical explanation is offered." W. S. REESE.

Tumors

FIBROMA OF THE SCLERA. V. CERCKEZ, Ann. d'ocul. 175: 390 (May) 1938.

Tumors of the sclera are rare, and fibromas are especially so. All authors class fibroma of the sclera in the group of orbital tumors.

V. M., a farmer aged 22, was seen because of exophthalmos of the right globe, with severe pain and loss of vision on that side. For the preceding two years he had had intermittent pain on the right side of the head. The pain grew gradually worse, and in the meantime he had been sent to a military hospital, where the Wassermann reaction was found to be negative, and roentgen examination of the orbit showed nothing abnormal. After four weeks of rest, the pain had improved, but a slight exophthalmos remained. The patient was returned to his regiment, but his condition grew worse, until he was unable to see well; the eye was then enucleated and examined pathologically. It showed a fibroma which came from the sclera.

Of particular importance is the pathogenesis, development and evolution of fibroma of the sclera. In this case the patient did not receive any trauma to the eye, which led the authors to believe that the tumor was of congenital origin. Extension of the tumor manifested itself behind in the orbit as well as in front, making it difficult for them to examine the back of the eye. The development and breaking down of the inflammatory phenomena were due probably to a special topography of the blood vessels, especially the veins. A bibliography accompanies the article. S. H. MCKEE.

VOLUMINOUS CHONDROMA OF THE LEFT ETHMOID REGION WITH MUCOCELES IN ALL SINUSES AND A GRAVE OCULAR SYNDROME. E. RUBALTELLI, Riv. oto-neuro-oftal. **14:** 48 (Jan.-Feb.) 1937.

A boy of 16 years, in good health, noted a progressive divergence of the left eye, protrusion of the globe and a marked reduction in vision. The disk was red and slightly elevated, with well defined edges and with markedly dilated and tortuous veins. Examination of the nose gave negative results. Palpation revealed a hard painless mass in the superomedial portion of the orbit. It was firmly fixed to the medial wall, from which it seemed to rise. Roentgenographically, an opaque mass could be demonstrated occupying the ethmoid cells, extending into the nasal fossa, the orbit and the maxillary sinus of the same side. In addition, the frontal and maxillary sinuses were clouded.

The mass was removed by a transmaxillary and central facial approach of Citelli. It had an osteocartilaginous consistency and histologically showed the characteristics of a chondroma. In addition, two large mucoceles were found occupying the frontal and the maxillary sinuses. Postoperative examination showed that vision, fundus and mobility had returned to normal. The text is illustrated with 9 photographs and drawings.

F. P. GUIDA.

TUMORS OF THE ORBIT: CYLINDROMA OF THE LACRIMAL GLAND. Q. DI MARZIO, Riv. oto-neuro-oftal. **14:** 65 (Jan.-Feb.) 1937.

Di Marzio reviews the literature on the subject and reports 7 cases of cylindroma of the lacrimal gland. He presents the clinical histories, operative procedures and results of histologic examination. Of these 7 patients, the first was well after sixteen years; the follow-up studies on the second and third were incomplete. The fourth patient showed a recurrence after twenty years and three years after a second operation died of cerebral metastasis. In the fifth case roentgen therapy was used after the third recurrence, with a cure of eight years' duration. The other 2 cases were recent.

After discussing the theories of the origin of these tumors, Di Marzio concludes that the elements which go to form the tumor vary, arising from the different tissues that go to make up the lacrimal gland embryologically; hence, the endothelial form of the growth at one time and the epithelial at another time. Early complete excision, including the lacrimal gland, followed by roentgen irradiation to avoid recurrence is advised, as the tumor is of a malignant nature.

F. P. GUIDA.

Vision

A VISUAL-ACUITY SURVEY. C. W. RAINEY, Am. J. Ophth. **20:** 930 (Sept.) 1937.

Rainey calls attention to the lack of a clinical report of a visual efficiency survey using the percentage method since the publication by the American Medical Association in 1925 of a standard method of

appraisal of loss of visual efficiency. He submits a survey of 721 men between the ages of 20 and 75 years, giving the following conclusions:

"1. Because the visual efficiency rapidly decreases with age, due to the loss of accommodation and the prevalence of subnormal and abnormal conditions of the eyes, the need for periodic visual-efficiency examination at one- to two-year intervals is apparent.

"2. Homatropine (4 percent) with cocaine (2 percent) can be used conveniently in determining the refractive state of the eyes at any age, without risk of inducing acute glaucoma.

"3. The use of 0.5-percent atropine solution, four drops a day for four days, is recommended to supplement the effect of homatropine in cases of high refractive error.

"4. Cylinder skiascopy, including the rotatory trial, was especially useful in groups 2 and 3, for patients whose vision could not be brought to normal and who were often unable to help in the subjective tests at the trial case.

"5. The manifest method of refraction is inadequate and inefficient as compared with the method using cycloplegia.

"6. The visual efficiency of each of the three groups could be increased by refraction and spectacles. The efficiency of the whole group was raised approximately 25 per cent for distance and 45 per-cent for near. The gain of the group already wearing spectacles was 14 percent for distance and 17 percent for near.

"7. The tests for color blindness were an aid to diagnosis.

"8. The ophthalmologist best serves the interests of the employee and employer.

"9. The Standard Method of Appraisal of Loss of Vision should be used as the basis of all group examinations.

"10. Examinations of the visual fields with the tangent curtain should be made in all cases in which the vision is not normal after refraction has been corrected."

W. S. REESE.

Sympathetic Ophthalmia

IS PERFORATION OF AN EYE NECESSARY FOR THE PRODUCTION OF SYMPATHETIC OPHTHALMIA? EINAR ANDERSSON, Acta ophth. **16:** 119, 1938.

There have been reported a number of cases of sympathetic ophthalmia in traumatized eyes wherein no penetrating wound has been found. The author makes the statement that in none of these cases has the eye been serially sectioned, which, he believes, is the only sure method by which to eliminate the possibility of the presence of a perforation.

In the case reported here, that of a 3 year old child, the history obtained from the parents was that of contusion by means of a stone to the right eye. No signs of perforation were evident clinically. Hemophthalmos and secondary glaucoma were present. Later, the cornea was stained with blood, and the eye became soft. Three and a half

VOLUMINOUS CHONDROMA OF THE LEFT ETHMOID REGION WITH MUCOCELES IN ALL SINUSES AND A GRAVE OCULAR SYNDROME. E. RUBALTELLI, Riv. oto-neuro-oftal. **14:** 48 (Jan.-Feb.) 1937.

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months after the injury a severe sympathetic ophthalmia occurred and progressed unremittingly despite all treatment, including enucleation of the exciting eye.

A minute needle-like penetrating wound involving the sclera, choroid and retina was found only after serial sections of the globe were made.

Although in this case the object which struck the eye was probably something other than a stone, the author reminds one that contusions may produce minute tears in the coats of the eye.

An interesting feature of the case was that the child did not react to tuberculin, nor could any evidence of tuberculosis be found on clinical or roentgen examination.

O. P. PERKINS.

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AMERICAN OPHTHALMOLOGICAL SOCIETY

FREDERICK H. VERHOEFF, M.D., *President*

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EUGENE M. BLAKE, M.D., *Secretary*

SHALL WE USE CYCLOPLEGICS? DR. W. H. CRISP, Denver.

Every refractionist should be able to make an approximately accurate measurement without the use of a cycloplegic. In many cases the difference between careful examinations without and with cycloplegia is slight, but there is always a risk of important differences being overlooked without cycloplegia. This is true even in cases of myopia, especially in the presence of astigmatism or of inequality of the two eyes. In cases of hyperopia careful use of bilateral fogging demonstrates that the amount of so-called latent hyperopia is less significant in most instances than is sometimes assumed. But cycloplegia is important for accuracy in estimating astigmatic error and in the exceptional cases of concealed hyperopia. As to astigmatism, the most important reason for the use of a cycloplegic is not its value in retinoscopic examination but rather the greater certainty of accuracy in the selection of cylindric strength and axis.

DISCUSSION

DR. GEORGE F. LIBBY, San Diego, Calif.: I use a 3 per cent solution of homatropine hydrobromate at about body temperature and apply it by raising the upper lid and putting a drop at the upper edge of the cornea. It is my custom to put 1 drop in each eye every five minutes until I have applied 6 drops, which gives satisfactory cycloplegia.

DR. W. B. LANCASTER, Boston: I agree with Dr. Crisp that there is a too general adoption of the idea that cycloplegia should be used in almost all cases, and I favor dispensing with it in many instances, even for children. One wishes to control accommodation because, by contraction and relaxation of the ciliary muscle, accommodation changes the refraction which one is trying to measure, increases myopia and decreases hypermetropia. Contraction of the ciliary muscle does not produce an astigmatic change in the lens so that it can correct an existing astigmatism; however, it is known that accommodation can neutralize astigmatism in some cases. It simply moves the whole conoid of Strum forward or backward, so that different portions of it fall on the retina. If the interfocal circle falls on the retina, all the lines will be equally distinct or equally slightly blurred, but accommodation does not neutralize the astigmatism by an unequal contraction of the ciliary muscle.

DR. H. S. GRADLE, Chicago: I do not agree with some of the statements made by Dr. Lancaster in regard to the lack of necessity for the use of cycloplegics in the majority of cases. It is true that refraction can be done adequately in a large percentage of cases without the use of a cycloplegic, but it is equally true that no eye can be examined thoroughly without its use. It is my belief that blindness could be prevented in a large percentage of cases of glaucoma by the early detection through the use of a cycloplegic.

DR. EDWARD JACKSON, Denver: The question of whether or not to use cycloplegics depends on whether the ophthalmologist wants to learn all that he can about the eye for which he is prescribing or whether he is content to follow his long experience and certain rules that have been laid down without finding out what might be learned about a particular eye or patient. It is always possible to learn more with the use of a cycloplegic than without. From the practical standpoint, the use of a cycloplegic often depends on the patient's resistance to having anything of that sort done.

Two things have often been overlooked in practice. First, a partial correction is not a correction of the error of refraction. The patient with a partial correction of myopia supplements this correction by looking obliquely through the lenses, thereby getting the effect of stronger lenses. Often he gets that effect at the cost of introducing astigmatism. A similar condition applies in cases of hyperopia.

One other point that is overlooked is that every person probably is able to relax his accommodation completely when he goes to sleep. He can learn to relax it completely and look through his glasses much more quickly than he can learn to use a lesser amount of correction and see clearly through his glasses. It is a general rule that all persons can relax accommodation when they go to sleep and that it is easier for them to learn to relax accommodation with the full correction before the eye than to relax it partially and still see clearly.

There are surprises all the time. There are children who take a + 1 diopter lens, and in spite of all efforts of the parents to have them wear glasses faithfully they will notice a definite blurring in reading the letters after wearing the glasses for some time. One must be guided in the individual case, but the patient is not being given a fair chance unless one tries to have him wear the full correction.

SUBJECTIVE STUDY OF VISUAL ABERRATIONS. DR. EDWARD JACKSON, Denver.

Newton explained the rainbow, and Helmholtz pointed out how the focusing power of the crystalline lens might change. But some common phenomena of vision still remain to be understood and explained. The conventional figure of a star, recognized for thousands of years, depends on aberrations presented in every dilated pupil. This can be studied by shutting off different parts of the pupil; this became known only when the practice of skiascopy was begun.

The radiating lines seen about the reflection of the sun from any polished surface are caused in the eye by reflections from the crystalline lens, near the anterior pole. They can be studied by looking at such

reflections through a small aperture. They probably assist the nutrition of the retina by distributing light to its pigment layer, without interfering with the usual retinal images. The lines are best seen against a black background. Subjective studies of the blindspot and visual fields, as described in the 1935 *Transactions of the American Ophthalmological Society*, have shown that certain primitive ocular movements are not coordinated with the retinal sense of direction and magnitude, which serves for most of the important functions of the eyes. The facts revealed by subjective study of vision are of practical value in diagnosis and in explaining to patients symptoms that have alarmed them.

DISCUSSION

Dr. W. B. Lancaster, Boston: All the rays that traverse the lens do not focus at a point, but in Strum's conoid there is a plane which can be selected by the eye for a good retinal image. (A diagram was used for purposes of demonstration.) The function of accommodation is to pick out the place which makes the best image, and that is what the eye does all the time. If the accommodation is paralyzed, the eye is unable to do this. With a mixture of rays the eye has a habit of picking out one place which suits it best, and when one introduces other rays at the periphery of the lens by dilating the pupil, the eye may still be able to select its original preferred point, as was mentioned by Dr. Crisp. The eye may do this in some instances, but if one wants to find out what the eye really does, one must examine it under the conditions under which it really works and not under cycloplegia, because then the aberrations are different and the eye must make a little different adjustment.

Mixed Cell Tumor of the Lacrimal Sac. Dr. Joseph L. McCool, San Francisco.

Mixed tumors occur commonly in the salivary, parotid and lacrimal glands and in the buccal mucosa. Mixed tumors of the lacrimal sac are rare. These neoplasms are of complex structure, usually presenting epithelial elements in the form of cell strands and neoplastic tissues, chiefly cartilage, mucous tissue and connective tissue. Any one of these elements may predominate, giving nearly pure chondromas, sarcomas or carcinomas, but usually all of the cell types are represented. Age appears to play no part, cases being reported in persons of from 11 months to 73 years of age.

A man, aged 54, complained of lacrimation of six months' duration and a slowly growing mass in the region of the left tear sac. Inspection revealed a hard, solid mass 1 by 1 by 0.5 cm. in the lacrimal fossa. The skin over the mass was normal in color and not particularly tense. The mass was not tender on palpation, and no secretion exuded through either punctum on pressure. The tumor was removed without difficulty. It was a soft, yellowish, translucent mass. Pathologic examination revealed at one edge of the section ciliated epithelium with marked round cell infiltration in the underlying tissues, which in places formed lymphoid nodules. Separated from this by a dense connective tissue septum was a mass of more neoplastic-looking tissue composed of a

mixture of fibroblastic tissue, round cells and cells of indefinite shape with rather large, clear, oval or round nuclei and without distinct nucleoli. Mitotic figures were common in some areas among the cells. Clear myxomatous areas were present in a small amount. There was infiltration of all the types of tissue between the striated muscle fibers at the edge.

A diagnosis of mixed tumor of the lacrimal sac was made. Radium was implanted in the cavity from which the tumor was removed. There was no recurrence ten months after the operation.

DISCUSSION

DR. JOHN W. BURKE, Washington, D. C.: A man, aged 73, seen on March 18, 1936, complained of sticking of the lids and epiphora. Examination showed a small growth protruding from the lower right punctum. Pressure on the lid would make the growth protrude further; on relaxation, it would drop back into the dilated canaliculus. On palpation, a rather firm, solid growth was felt. On April 23, with local anesthesia, the canaliculus was split to the inner canthus. The growth was lifted out. It did not seem to be adherent, and there was no bleeding when it was removed. The mucous membrane of the canaliculus was stretched and slightly hyperemic but otherwise showed no changes. The tumor was pear shaped and measured 8 by 5 mm. It was pinkish yellow, slightly vascular and not pigmented.

Microscopic examination showed a papillary tumor with small connective tissue cores covered by multiple layers of stratified squamous epithelium with some leukocytic infiltration in the tumor and in the stalks. There was no evidence of infiltration, and the lesion had all the appearance of a benign tumor. A diagnosis of epithelial papilloma was made.

PANOPHTHALMITIS AND SYMPATHETIC OPHTHALMIA. DR. BERNARD SAMUELS, New York.

This article appeared in full in the November issue of the ARCHIVES, page 804.

METASTATIC SEPTIC ENDOPHTHALMITIS WITH RING ABSCESS OF THE CORNEA: REPORT OF A CASE WITH DESCRIPTION OF ANATOMIC CHANGES. DR. C. W. TOOKER, St. Louis.

A man, aged 53, entered the hospital in June 1932 for the treatment of a bilateral mastoiditis. After operation he seemed to have made a good recovery but was brought to the hospital again in January 1933, when a staphylococcic septicemia was discovered. Vision in the left eye was limited to perception of light. The cornea was clouded. The iris and anterior chamber were filled with exudate. Blood cultures on two occasions showed the presence of staphylococci. The patient lived three days, and a complete autopsy was done. The left eye was removed and sectioned.

(Sections and photomicrographs were shown to demonstrate the anatomic changes.)

ORBITAL HYPEROSTOSIS: ITS OCCURRENCE IN TWO CASES OF MENINGIOMA OF THE SKULL. DR. ARNOLD KNAPP, New York.

This article appears in full, with discussion, in this issue of the ARCHIVES, page 996.

(*a*) LEIOMYOMA AND (*b*) BLOOD CYST OF THE IRIS: REPORT OF CASES. DR. E. C. ELLETT, Memphis, Tenn.

This article will appear in full, with discussion, in a later issue of the ARCHIVES.

OCULAR MANIFESTATIONS IN BRUCELLOSIS (UNDULANT FEVER). DR. JOHN GREEN, St. Louis.

This article will appear in full, with discussion, in a later issue of the ARCHIVES.

SYPHILITIC OPTOCHIASMIC ARACHNOIDITIS. DR. DERRICK VAIL, Cincinnati.

A review of the literature revealed numerous reports of cases of optochiasmic arachnoiditis in persons who showed clinical or laboratory evidence of syphilis. A man, aged 58, who was thought to have been tabetic for five years and had received long courses of antisyphilitic treatment, complained of progressive loss of vision in spite of the treatment. The right eye became entirely blind, and vision in the left eye was reduced to ability to count fingers at 3 feet (91 cm.) because of a large central scotoma. The outline of the disks was sharp, except for nasal blurring and a depressed center, and there was a visible, greenish white lamina cribrosa; the vessels were markedly reduced in caliber. A diagnosis of optochiasmic arachnoiditis was made. Craniotomy in the right frontal region, performed on April 4, 1938, by Dr. Nolan Carter, of Cincinnati, disclosed an old leptomeningitis, involving especially the under surface of the frontal lobes. The chiasm and optic nerves were hidden and entangled in sheetlike bands of old inflammatory tissue. Examination of a fragment of the thin inflammatory membrane showed a thickened arachnoid with epithelioid cells and secondary fibrosis.

DISCUSSION

DR. RALPH I. LLOYD, Brooklyn: I had a case of specific chiasmic arachnoiditis in which the change came on suddenly. The right eye became blind overnight. Examination of the other eye showed temporal quadranopia. The patient was seen by an otolaryngologist, who removed the ethmoid cells on the affected side. The tissue was sent to a pathologist, who reported hyperplastic rhinitis and said that it was a sufficient cause to explain the sudden loss of vision. In about a month the other quarter of the affected half-field disappeared, leaving a picture typical of that seen in disease of the pituitary gland, i. e., one blind eye and hemianopia in the other eye. The vision before the operation was nearly 20/20, and the knee jerks were normal. At the end of the following month vision in the macular area of the remaining half-field was com-

pletely gone. In the course of the next three months the patient was totally blind. His Wassermann reaction was 4 plus. He first said that he had never had a syphilitic infection, but he finally admitted having had one thirty-five years previously.

(The manner in which the visual field became constricted to the vanishing point was demonstrated by slides, which revealed particularly the similarity of the changes in the visual field to those associated with lesions of the pituitary body.)

Dr. Derrick Vail, Cincinnati: I am grateful to Dr. Lloyd for pointing out the progressive loss of the field of vision in these cases from beginning to end. He pointed out the importance of the chiasmal area in certain lesions and called attention to chiasmic defects in the visual field, which can explain many other ramifications, for example, the traumatic type of optochiasmic arachnoiditis and the suggestion that syphilitic atrophy of the optic nerve is due to a lesion arising in the chiasmic cisterna and affecting the optic nerve.

Lesion of the Optic Tract Probably the Result of Infected Sphenoid Sinuses. Dr. Albert N. Lemoine, Kansas City, Mo.

This article appears in full, with discussion, in this issue of the Archives, page 966.

Correction of Entropion by Transplantation of the Orbicularis Muscle of the Lower Eyelid. Dr. John M. Wheeler, New York.

Many procedures have been suggested for the correction of spastic entropion. Most of them, but not all, depend for their effectiveness on a downward pull, calculated to unroll the inverted lower lid and to prevent turning in of the margin by downward traction on the skin.

The operation described depends for its effect on pressure at the lower margin of the tarsus of the lower lid, obtained by tightening some of the fibers of the orbicularis muscle. The operation prevents entropion and at the same time gives support to the eyelid.

DISCUSSION

Dr. Edward Jackson, Denver: Is there any difficulty in isolating the portion of orbicularis muscle that is wanted, and how does one judge this portion in cutting the strips of muscle before proceeding to use them?

Dr. J. E. Weeks, Portland, Ore.: For many years I have employed a simple operation for the correction of spasmodic entropion of the lower eyelid with most satisfactory results. An incision is made 3 mm. below and parallel to the margin of the lid, extending from the punctum almost to the outer commissure. A curved incision is now made connecting with the extremities of the first incision, including as much integument below the first incision as is thought desirable. The subcutaneous tissue and the underlying fibers of the orbicularis muscle are excised with the skin. The margins of the wound are approximated by means of fine silk sutures. Three sutures are then passed through the margin of the

lid from above downward, beginning in the intermarginal space and emerging on the cutaneous surface 2 mm. below, one suture being in the middle of the lid and one on each side, about halfway to the canthi. Each suture is then passed into the skin of the cheek about 4 mm. directly below its point of first emergence, again emerging 2 mm. farther down. A small roll (3 mm. in diameter) of iodoform gauze is passed beneath the sutures, and the ends of the sutures are tied, causing marked ectropion of the margin of the lower lid. The sutures are removed at the end of six or seven days.

DR. RAMON CASTROVIEJO, New York: With no knowledge of Dr. Wheeler's operation but simply after watching him work I decided to operate in a couple of cases. I did not find the procedure difficult. I have also been able to correct entropion of the upper lid by the same method.

DR. JOHN M. WHEELER, New York: Procaine hydrochloride is thoroughly injected into the lower lid, so as to get good magnification of the fibers and to reduce the entropion while the operation is going on. In the second procedure I spoke of it is perfectly feasible to clamp the lid so that there will be no hemorrhage. Since one is almost sure to cut the palpebral arteries during dissection, one should clamp them. I like to leave the orbicularis muscle in place and with the scissors cut down against the tarsus in which the strips are outlined; I then lift the outlined strips with a pair of forceps and cut under them with very fine scissors. By holding the strip on a hook it is easy to carry on the dissection toward the end of the lid and then to put in the sutures in such a way as to be sure of the accurate overlapping and the position of the tightened strips of orbicularis muscle.

A NEW METHOD FOR TRANSPLANTING PTERYGIUM. DR. EDWIN M. NEHER, Salt Lake City.

This article will appear in full, with discussion, in a later issue of the ARCHIVES.

INFERIOR IRIDOTOMY IN OPERATIONS FOR CATARACT ON EYES WITH POSTERIOR SYNECHIAE OR PUPILLARY MEMBRANE: VALUE OF OPERATION. DR. PAUL A. CHANDLER, Boston.

This article appeared in full in the October issue of the ARCHIVES, page 641.

DISCUSSION

DR. DERRICK VAIL, Cincinnati: Dr. Chandler's contribution is going to be of great value to any one who does operations for cataract, and I know that I will follow his precepts in such cases as he described. For a number of years I have performed inferior iridotomy in other cases, particularly in those in which there has been loss of vitreous following the extraction of cataract. After loss of vitreous the pupil always assumes a hammock shape or a boat shape. This produces a drag on the pillars of both sides, which may be obviated by doing an inferior iridotomy at the time of the extraction, in spite of the danger of further loss of vitreous. If the operation is skilfully done with sharp Noyes scissors, further loss of vitreous is not great.

OPHTHALMOLOGY COMES OF AGE: I. THE YEARS 1908-1915, UP TO THE ESTABLISHMENT OF THE EXAMINING BOARD. DR. SYLVESTER JUDD BEACH, Portland, Me.

History relates that in 1913 the three national ophthalmologic societies appointed a joint committee. After due consideration, this committee evolved the scheme of an examining board. As a consequence, in 1915 what is now the American Board of Ophthalmology was organized. Even in the chronicles of the board there is no hint that there was anything more complicated than this about the origin of a movement which has revolutionized postgraduate medical education. In reality, for a period of years several groups had in divergent ways been wrestling with an unsavory situation. It was by deft use of the momentum they created that this solution was finally reached.

DISCUSSION

DR. GEORGE F. LIBBY, San Diego, Calif.: In 1912, under Dr. Jackson's directions, there was established in Denver a postgraduate school for advanced teaching in ophthalmology. There were 11 students, 1 from Pennsylvania, 2 from the Middle West and 8 from Denver. The course was of six weeks' duration. In the following year, after a year's reading in ophthalmology under Dr. Jackson's direction, 3 members of the class received the degree of Doctor of Ophthalmology at the state university. This movement, leading to the certification of men who had taken advanced work in ophthalmology, such as the course given in Pennsylvania and similar courses in the University of Minnesota, and to the degree of Doctor of Science in Ophthalmology, had an important bearing on the development of the board.

DR. W. B. LANCASTER, Boston: The society is grateful to Dr. Beach for this important historical contribution. Perhaps the ophthalmologists here think that it is a simple matter to write such a paper; that all one has to do is to look up the records of the *Transactions* to find what was done year by year! There is a great deal more to it than that. The historian has to evaluate what he finds and interpret it, but even the records are meager. They simply say "it was moved and carried, etc." Take the epoch-making meeting of last night; when the future historian tries to find out the facts he will simply be confronted with the record "moved and voted." Nothing will be said of how Horatio held the bridge; who fought on his right hand and who on his left hand; what soldiers stood behind him or how they met the onslaughts of the reactionaries and the die-hards! Perhaps every one thinks that when the American Board of Ophthalmology was formed everything was peaceful, but I can assure them that that baby was born with tears and travail and that when the board first made its appearance many a finger of scorn and ridicule was pointed at it by men in high position. Fortunately in those days the leaders were men of vision and of courage, but of vision especially, who could see the future when others were in the fog and who led on to great achievement, so that now ophthalmologists are proud to have the stone which was originated by many of these men made the headstone of the corner: ophthalmology leading all the specialties, from otolaryngology to surgery.

TRAUMATIC RETINAL ANGIOPATHY. DR. ARTHUR J. BEDELL, Albany, N. Y.

The change in the fundus in cases of traumatic retinal angiopathy, consisting of large fluffy white masses with many superficial and deep hemorrhages about the disk, is pathognomonic. This condition was mentioned by Jacobi in 1868 and was completely described by Purtscher in 1910, since which time his name has been commonly associated with it.

(Lantern slides of previously published illustrations accompanied a review of the literature.)

I wish to add 3 new cases to the small number reported. The condition in 1 was caused by a severe injury to the side of the head; in another, by compression of the chest, traumatic asphyxia and fracture of the dorsal vertebrae, and in the third, by fracture of the third and fourth lumbar vertebrae.

(Serial color photographs of the fundus were shown to demonstrate the clinical history.)

THE CULTIVATION OF HUMAN CONJUNCTIVAL AND CORNEAL EPITHELIUM IN VITRO. DR. PHILLIPS THYGESON, New York.

Certain viruses affecting the human conjunctiva and cornea have failed to grow in cultures of chick embryo tissue, and many require human epithelial tissues. Experiments on the cultivation of human conjunctival and corneal epithelium are reported, including notes on the histologic structure of the developing tissues. A technic suitable for use in virus studies is outlined.

DISCUSSION

DR. DANIEL B. KIRBY, New York: Dr. Thygeson is to be congratulated on the development of this method of cultivation of tissues from the human eye. Dr. Carrel and Dr. Ebeling have both expressed themselves as admiring very much the progress which Dr. Thygeson had made in this research. It will be of further value in researches in psychology and in pathologic studies of other human ocular tissues.

DR. RAMON CASTROVIEJO, New York: I was particularly interested in Dr. Thygeson's presentation, because he has brought out some points that had been the object of debate. He has illustrated that epithelial tissue cultivated in vitro gives origin only to epithelial tissue. In the same way, connective tissue can give origin only to connective tissue cells.

In reviewing the literature on the histologic picture of keratoplasty, published by some French authors, I was surprised to find the statement that connective tissue cells which developed to repair corneal wounds originated from epithelial cells. This observation entirely disagrees with Dr. Thygeson's findings and with my own microscopic study of corneal grafts both in animals and in human beings. I found, like Dr. Thygeson, that epithelial cells can reproduce only epithelial cells and that connective tissue cells were produced by preexisting cells of the same nature.

Another point stressed by Dr. Thygeson in his presentation was that the growth of tissue cultures was the same, whether the tissue was obtained from fetuses, stillborn infants or adults. This observation also disagrees with the clinical finding that transplants obtained from stillborn infants behaved in exactly the same manner as transplants obtained from adults' eyes.

Dr. Thygeson deserves to be congratulated for his thorough and most interesting presentation.

RESULTS OF AUTOTRANSPLANTATION OF CORNEA INTO ANTERIOR CHAMBER: THEIR SIGNIFICANCE REGARDING CORNEAL NUTRITION. DR. TRYGVE GUNDERSEN, Boston.

This article appeared in full in the October issue of the ARCHIVES, page 645.

SOME PHARMACOLOGIC REACTIONS OF ISOLATED IRIS MUSCLES. DR. PARKER HEATH, Detroit.

An experimental study was made of some of the actions of iris dilators and sphincters in animals. The method used consisted of a delicate suspension of isolated sphincter or dilator in solution; the changes were electrically amplified and then recorded by light beams on sensitized paper. Various stimuli and depressors were used, especially those classed as sympathetic and parasympathetic. Physiologic or spontaneous reactions were noted.

DISCUSSION

DR. H. S. GRADLE, Chicago: About twelve years ago I worked on the time reaction of the pupil. I employed a similar method in the human being in the hope of establishing a more accurate timing of the pupillary reaction. The apparatus employed was a modification of the ordinary clinical electrocardiograph, and the method was similar to that described by Dr. Heath. Interestingly enough, the electrodes were applied to both sides of the sclera (the difference in potential was what was desired), and the results were negative. One can obtain satisfactory graphs on the human subject by the use of nonpolarizing electrodes, which show clearly the electrical reactions of contraction of the sphincter and also of the dilator fibers. It is true that the stimulus used here was merely the stimulus of light, but different values for the sphincter and dilator effects were obtained which led to the impression that the dilator fibers are infinitely weaker than the sphincter. The experimental method described by Dr. Heath is applicable to the living human eye, with the exception that side currents are set up which form almost uncontrollable reactions of the extraocular muscles which tend to cloud the issue. Consequently, Dr. Heath's method is a marked advance in using the isolated iris muscle and in eliminating all extraneous influences.

ANISEIKONIA. DR. WALTER B. LANCASTER, Boston.

This article appears in full in this issue of the ARCHIVES, page 907.

ANISEIKONIA: A CLINICAL STUDY OF 836 PATIENTS. DR. CONRAD BERENS, New York.

The data obtained in this study seem to indicate that correction of aniseikonia may be a factor in some cases in the relief of ocular complaints and general symptoms which apparently are not eliminated by the wearing of ordinary correcting lenses, orthoptic training and other forms of treatment. The correction for ametropia was changed in some cases; in others, accommodation seemed to be improved for some unknown reason. Muscle balance was undoubtedly changed in some instances, and fluctuations in the physical condition may have been coincident with the wearing of the iseikonic lenses; these and other factors should be taken into consideration in evaluating results.

DISCUSSION ON PAPERS BY DRS. LANCASTER AND BERENS

DR. EDWARD JACKSON, Denver: Studies on aniseikonia as reported in the papers of Drs. Lancaster and Berens have far-reaching effects. They are close to the question of cerebral action and association, beyond the boundaries of what the great mass of people have learned to do with their eyes. Individual differences must be considered before the significance of the tables that were presented is understood.

DR. HERMAN N. BURIAN, Hanover, N. D.: I have studied the importance of peripheral fusional stimuli for the relative position of the two eyes, for the process of fusion is in itself a well rounded problem of physiologic optics, and its investigation was not undertaken with specific regard to aniseikonia. As a side result, however, I obtained, as Dr. Lancaster pointed out, the conclusive proof that the measurements of artificially induced differences in size are identical, no matter whether they are taken with or without ocular movements. In other words, an actual difference in size was measured, not a possible anisophoria.

One has to assume that the relative difference in the sizes of the ocular images gives rise to difficulties in fusing the images of the two eyes. The result is that in persons affected with aniseikonia the constant struggle to bring about and maintain fusion produces the well known symptoms. One has to rely on the reports of the patients as to the relief they obtained by wearing an aniseikonic correction and from these reports to draw conclusions concerning the validity of one's assumption of the effectiveness of size corrections. There is, however, an objective sign which seems to me especially convincing as to the value of aniseikonic corrections for the achievement of perfect binocular vision. This is a fact that a marked improvement of depth perception can be obtained by the wearing of an aniseikonic correction. A number of intelligent patients who are good observers report spontaneously that they have noticed such an improvement or that they have experienced for the first time real stereoscopic three-dimensional vision. They often report in connection with this a considerable improvement in vision for fine, close work and for certain outdoor activities.

Objectively, this subjective impression of the patients has been checked with the Keystone chart, and it has been found that there actually was a marked improvement in their depth perception. If such patients do not wear their glasses for a few hours, their depth perception will be more or less reduced. Immediately after putting on the glasses, they show 100 per cent depth perception and do not lose it for a longer or shorter time after taking the glasses off.

Finally, I should like to mention a last point. Dr. Berens has mentioned that in his cases there was no noticeable influence of aniseikonic corrections on the phorias of the patients. This is not astonishing, and it is evident that one cannot expect such an influence if one defines the phorias as the position of rest of the eyes. This position, so far as it is due to mechanical factors, cannot be expected to change. Under normal conditions of seeing it is not true that all innervational influences are excluded, and one can well imagine that it will be much easier for a patient with a considerable amount of phoria to overcome the anomaly in the position of rest if the process of fusion is facilitated by matching the images of the two eyes. This is probably another important factor which contributes to the great benefit some patients derive from the wearing of aniseikonic corrections.

ALEUKEMIC LYMPHOSIS INVOLVING THE UPPER LIDS: PATHOLOGIC CHANGES. DR. FREDERICK T. TOOKE, Montreal, Canada.

The patient, an elderly, corpulent person, had heavy pendulous eyelids that were everted with difficulty. The palpebral conjunctiva was ischemic, appearing somewhat like moist blotting paper. The skin was not attached to what felt like a generalized involvement of the meibomian glands. The lids measured from 7 to 7.5 mm. A contrast measurement of a patient of the same body weight was 2 mm.

Clinical examination revealed a generalized glandular involvement, including the posterior cervical glands, the preauricular glands, the epitrochlear glands and the axillary and inguinal glands. Adiposity precluded palpation of the spleen. Biopsy was performed on the left upper lid and on one cervical gland.

Microscopic sections of lymph gland showed loss of normal structure due to an intense proliferation of closely packed, small, round lymphoid cells. The cells were uniform, tending to be a trifle angular in some areas with a suggestion of clinging to the reticulum. The picture presented corresponded to that found on the borderline between a lymphatic leukemia and a relatively mature form of lymphosarcomatosis. One is inclined to classify the condition among the kataplasias, i. e., as a rather immature form of lymphatic leukemia.

Sections from the eyelid showed densely packed lymphoid cells which were of medium size; they exhibited occasional mitotic figures and corresponded to the elements met with in the lymph gland described here. One also places the proliferation among the more immature forms of kataplastic processes, with an appreciation of the fact that it borders on a lymphosarcomatosis. The pathologic changes were similar to, though somewhat more immature than, those frequently described under the term "lymphatic leukaemia cutis."

The blood picture showed marked anemia, with a tendency for the cells to be somewhat larger than normal and of poor regenerative ability. The white blood cells showed a deficiency in the number of myeloid elements, with some immature forms. The lymphocytes were increased, and this increase appeared to be due to the presence of occasional lymphoblastic cells, similar to those met with in the lymph glands.

The blood picture was not typical of lymphatic leukemia or of lymphosarcomatosis. One is inclined to believe that the condition probably lies on the borderline between these two diseases. It is understandable that such an intermediate type might well occur when one considers the intimate relationship between these two conditions and the fact that the latter is simply a pathologic process of a more immature undifferentiated nature. For the purpose of classification the condition described here is probably best considered aleukemic lymphosis.

DISCUSSION

Dr. John E. Weeks, Portland, Ore.: Could the condition in this case be Mikulicz' disease, as described by Professor Mikulicz, of Breslau, Germany, some years ago?

Dr. Edward Jackson, Denver: Although radiation therapy has been tried on the lymph glands in cases such as this, I should try the effect of vitamins, especially vitamin B.

Dr. John E. Weeks, Portland, Ore.: A case similar to that reported by Dr. Tooke came under my observation a year and a half ago. I was surprised at the enlargement of the glandular elements and particularly of the eyelids themselves. Under irradiation the lymphomas entirely disappeared or were reduced in size at least during a period of about nine months, and the patient's general health appeared to improve.

Dr. Frederick T. Tooke, Montreal, Canada: In reply to Dr. Weeks' question, I thought that the pathologic changes in Mikulicz' disease were much more definitely sarcomatous than those manifest in this case; also the blood picture as described here should answer Dr. Weeks' question more clearly.

The suggestion of Dr. Jackson regarding treatment might have been tried, but probably it would not have had much effect, considering the rapid decline of the patient from the time he came under observation.

Orbital Mechanics and Limitations and Movement of the Eye, Optic Nerve, Oblique Muscle and Müller's Muscle with Relation to Exophthalmos, Separation of the Retina, Thrombosis and Postoperative Malaise. Dr. Clifford B. Walker, Los Angeles.

Several problems are encountered in work on the separated retina, the more notable of which follow: (1) absolute blindness immediately after traumatic separation which persists without interruption and (2) nausea and vomiting, apparently of the cerebral type, following more or less difficult operations in certain quadrants, with perhaps excessive tension on the optic nerve in spite of the fact that tenotomy is not performed.

Some of the factors involved, together with a consideration of the accessory function of the smooth and oblique muscles, deduced from a common type of retinal separation with the tear in the neighborhood of the attachment of the superior oblique muscle, can be studied to advantage in the light of orbital mechanics exhibited by models. Visualization of these mechanics has been found inefficient with large heavy models used almost exclusively heretofore. An attempt has been made, therefore, to make skeletonized or semitransparent models of exact size which are differentiated with color and enlarged by means of photoprojection and have various degrees of motion and tint.

Book Reviews

Methodik des optischen Raumsinnes und der Augenbewegungen. Abderhalden's Handbuch der biologischen Arbeitsmethoden. By Armin Tschermak-Seysenegg. Section V, part 6, no. 10. Pp. 325, with 88 illustrations. Berlin: Urban and Schwarzenberg, 1937.

Since F. B. Hofmann's contribution to Tigerstedt's "Handbuch der physiologischen Methodik" (1914), no comprehensive work concerning the methods used in the investigation of the sense of space and of the ocular movements has been published. This void is now filled by Armin Tschermak's exhaustive article, which is all the more welcome since the work of E. Hering has been developed and continued by this author more than by any other living physiologist. Nobody can compete with Tschermak's enormous theoretic knowledge and practical experience in the field under discussion. This also accounts for the facts that in his article theoretic discussions take up more space than one would expect to find in a book dedicated to the methods of investigation and that it gives in detail particularly the methods used and developed by that author himself, although all the other current methods are mentioned.

In an introductory chapter Tschermak gives a brief recapitulation of the basic ideas of what he has termed the exact subjectivism in the physiology of the senses, emphasizing the necessity of strict discrimination between the objective geometric distribution and the subjective impression of the position in space.

The first part of the work deals with the general methods in the investigation of the sense of space, the fixation of the head, the direction of gaze of the observer, etc. The author emphasizes especially the great advantage of self-recording devices, with the help of which, particularly of an appropriate punch, permanent records of the measurements can be obtained on paper without the necessity of reading the scales after each setting. A special chapter is devoted to the mathematical method of the theory of probabilities.

The special discussion of the sense of space is subdivided into the methods devised for the investigation of the perception of space in monocular and in binocular vision. To the first pertain the methods of determining the visual field; these methods are not treated exhaustively, since they have been considered in detail in other, mostly clinical, publications. It is, however, interesting to note that Tschermak reemphasizes the fact that the restriction of the color sense in indirect vision is purely relative and that one is not warranted in speaking of absolute color blindness in the periphery of the visual field. The relative localization in space without movements of the eyes is discussed more exhaustively. The theoretic significance of the various asymmetries in localization (angular discrepancies, discrepancies in direction, Kundt's partition experiment, the radial shrinkage of a test object in indirect vision, etc.), which play such an important role in the foundation of the subjectivistic conception of physiologic optics, is treated thoroughly, and the methods for their

determination are described. Tschermak's deductions from these discrepancies concerning a difference between the nodal projection center of the retina and the prenodal functional center as point of reference for a division of the retina are worthy of consideration. He emphasizes that the significance of the discepancies in question can be evaluated properly only when it has been cleared up whether, and to what extent, they depend on asymmetries of the dioptric apparatus of the eyes.

The examination of the sensorial and motor orientation in space, i. e., the absolute and egocentric localization, disclination and conclination of the vertical and the horizontal meridian with various positions of the head and body and under the influence of acceleration, is discussed at length. Much space is taken up by interesting theoretic considerations, which are warranted because of their importance for the physiology of flying and for clinical examinations.

The methods concerned with the investigation of binocular space perception are subdivided into a number of chapters. The first chapter deals with the study of the haploscopic and stereoscopic effects produced without the use of instruments. The conception of the sensorial cyclopean eye is discussed and explained with the aid of a diagram. Of special importance for the clinician is the method wherein haploscopic and stereoscopic effects are achieved with the aid of colored (red and green) devices, the method of Haidinger's polarization brushes and, above all, the after-image method. The last method becomes especially important in the investigation of the visual act of squint, because it permits the determination of the retinal (sensorial) correspondence. Methods are described for measuring the angle of squint and examining the dominance of one eye by means of retinal rivalry.

A detailed description is given of the mirror haploscope, especially in the form developed by the author.

The methods for determining the corresponding retinal points, particularly those based on the determination of the apparent frontal plane ("horopter determinations"), are treated at length, and the instruments and their use are described. The methods based on other criteria (single appearance of a test object, constancy of the visual directions and maximal sensitivity to stereoscopic differences) are also taken into consideration. In this chapter mention is made of the influence which differences in size and shape of the ocular images (aniseikonia) have on the form of the horopter. The last chapter of the methods concerned with space perception deals with binocular depth perception. After an introduction giving the basic ideas concerning depth perception, there is a description of a number of arrangements and instruments for the investigation of this function. The various factors which influence stereopsis and have a bearing on the stereoscopic methods—time factor, factors concerning the localization of the images, dioptric factors and oculomotor factors—are discussed and their importance evaluated. The chapter on binocular vision closes with a discussion of secondary, empiric and interpretive movements which lead to the sensation of depth not based on vision with disparate retinal points.

The next part of the work deals with the investigation of movements of the eyes. The question of the center of rotation of the eyes and of its determination on the axes of rotation as well as characteristics

of the ocular muscles and the laws governing the movements of the single eye are discussed in detail. The special methods for testing the laws of Donders and Listing are presented, and particular emphasis is placed on the after-image method which Tschermak considers best and, in general, quite adequate. An exhaustive description is given of Tschermak's model illustrating Listing's law. For the examination of the voluntary movements of the eye, as they occur under the ordinary conditions of seeing, one has to resort to one of the various methods of photographic registration, which are described in full.

The discussion of the associated movements of the two eyes is begun with Hering's law of equal innervation of the oculomotor apparatus. This is followed by a discussion of the complex of accommodation, convergence and pupillary reaction, i. e., the adaptation of the eyes for near and for distant vision. Special emphasis is given to those movements which Tschermak designates as "adaptative movements." They bring the eyes comparatively slowly into a suitable tonic equilibrium, in which they remain for a certain length of time. These movements are: (1) corrective movements, i. e., movements which tend to overcome differences in the oculomotor apparatus of the two eyes (heterophorias); (2) fusion movements, which are produced under artificial conditions (prisms, etc.) and serve the purpose of maintaining binocular single vision; and (3) static changes in the position of the eyes, the purpose of which is to maintain the normal position of the meridians of the eyes in spite of the influence which changes of the position of head or body have on the oculomotor apparatus.

For the investigation of the heterophorias, Tschermak describes, besides mentioning other methods, his own phorometer and his after-image method, which allows the determination of heterophorias with purely monocular fixation. The methods for investigating the fusion movements (with the haploscope or prisms) and the static and optokinetic changes in the position of the eyes are described briefly.

The work closes with a discussion of cinematographic devices, i. e., devices with the aid of which sensation of movement can be produced (stroboscope, strobostereoscope, moving pictures, Archimedes' spiral, Dvorak-Nach's phenomenon, Pulfrich's stereo-effect, etc.), and with a short mention of the methods for the production of optokinetic nystagmus.

In the scope of a short review it is impossible to do justice to Tschermak's work, with its abundance of knowledge, facts and valuable suggestions. Whoever is interested, or wants to do research work, in the field of space perception and the physiology of ocular movements will have to refer to Tschermak's work in the original.

A. BIELSCHOWSKY.

Ottalmologia dei paesi caldi. By Prof. Vittoria Ruata, Italy. Cloth. Price, 45 lire. Pp. 362, with 92 illustrations and 4 colored plates. Milano: Ulrico Hoepli, 1938.

Vittorio Ruata, an associate at the Royal Eye Institute of the University of Rome, has written a concise, practical, theoretic volume of moderate size on tropical ophthalmology. Ruata has had the opportunity

of living and practicing in North Africa (Libya and Egypt) and of studying in other tropical countries. In the preface he states that he was urged to undertake this task because of the lack of a similar manual, not only in the Italian medical literature but in that of some other countries as well.

The author adheres strictly to his purpose—to be practical—referring to experimental theories only when he thinks that they may be of some help to the reader.

The volume is divided into nine chapters. In the first chapter Ruata describes the effects of climatic and atmospheric elements—wind, dust, light, etc.—on the eye in tropical countries, their consequences and their causal relationship to diseases of the conjunctiva or to the other structures of the eyeball.

After a review of the lesions caused by insects or by animal bites, the animal parasites which frequently cause ocular disease are listed.

As would be expected, the chapter on trachoma is the pièce de résistance of the book. Ruata has had a great deal of experience with this problem in European countries and in Egypt, in which, he asserts, trachoma affects a greater percentage of the population than in other countries. The reader will find of interest the observation of the author that in Tunisia, Libya, Egypt and Ethiopia the black race is less affected by trachoma than the yellow or Mongolian race. While the native Egyptian population is affected nearly 100 per cent, as one goes into the Egyptian Sudan (where the population is mostly Negro) trachoma is found in only about 10 per cent of the population. Also, in Ethiopia and in Somaliland trachoma is almost absent among the natives, while on the opposite side of the Red Sea (where the population is mostly of Arabic blood) trachoma is found in almost 100 per cent of the population. For these reasons the author thinks that race rather than climate is the principal factor determining the incidence of trachoma in the tropics.

The last few chapters of the book are devoted to tropical diseases which affect the ocular adnexae.

In my opinion the author has succeeded in presenting a practical volume on tropical ophthalmology which should be a worthy additon to the library of any student of ophthalmology, whether or not he is practicing in tropical countries.

G. BONACCOLTO.

An Introduction to Clinical Perimetry. By H. M. Traquair, M.D., Edinburgh. Third edition, revised and enlarged. Price, 30 shillings. Pp. 320, with 227 illustrations and 3 colored plates. London: Henry Kimpton, 1938.

Dr. Traquair's textbook on perimetry, which has become a classic, now appears in a third edition. In a foreword by Norman M. Dott, the neurosurgeon, the value of quantitative perimetry is emphasized, and it is pointed out that perimetry has now become an integral part of neurologic investigation, that speed of examination is particularly essential and that fatigue of the patient must always be reckoned with. The book is revised and enlarged, with 48 new illustrations, and new advances in this field are recorded. Simplicity of expression and the

demonstration of essentially practical methods remain outstanding features. It is one of Dr. Traquair's great merits that the procedures which he advocates are simple, both as to instrument and as to technic, and within the reach of all and that it is not the elaboration of apparatus that is important but the understanding of the one who takes the field. Emphasis is justly laid on the interpretation and evaluation of the findings. The author believes that it is unnecessary to burden clinical procedures with elaborateness of doubtful practical value. Interpretation depends on knowledge of the anatomy of the visual paths and of the various ways in which different pathologic conditions act. Finally, as the examination is subjective, the psychic conditions which may be of influence must be recognized.

Ophthalmologists are indebted to Dr. Traquair not only for a splendid work but for the admirable manner in which he has presented a difficult subject.

ARNOLD KNAPP.

An Introduction to Clinical Scotometry. By John N. Evans, M.D., F.A.C.S. Cloth. Price, $4. Pp. 266, with 57 illustrations, appendixes and bibliography index. New Haven, Conn.: Yale University Press, 1938.

In the first six chapters of this book the author gives in detail the history of angioscotometry, a description of the equipment and the technic. In the remaining eight chapters he describes in detail the normal angioscotoma and compares it with angioscotomas found in primary ocular disease and with those associated with lesions of the nerve bundles. The appendixes contain much important data.

The author believes that an angioscotoma is not the actual measurement of vessel shadows but is a measure of the perivascular space. A hypothesis has been devised correlating the angioscotoma with the widening of the perivascular space which produces a dysfunction of the synapses in the retina due to oxygen deprivation. This theory is supported clinically in that although the retinal arterioles are narrowed by pressure on the globe, the angioscotoma becomes wider.

Angioscotomas produced by retinal edema, diseases of the nasal accessory sinuses, glaucoma, true neuritis and pseudoneuritis, intracranial vascular lesions, complicated pregnancy and retinal arteriosclerosis and those associated with strabismus are differentiated from those produced by variations in the normal eye.

As all defects of the visual field cannot be explained adequately by the angiogenic (perivascular space) hypothesis, the neurogenic (nerve fiber bundle) hypothesis has been considered and the respective defects differentiated. The author feels "that wedge-shaped defects with the apex pointing toward the blind spot are ocular and angiopathic in origin, but where the apex is toward the macula the lesion is primarily in the pathway. The picture is rendered atypical, however, where increased intracranial pressure is superimposed." This apparent argumentative point increases the value of the book, which is indispensable to the ophthalmologist who does not relegate the examinations of the visual fields to his secretary or office nurse.

The subject is presented completely in this book and is a challenge to ophthalmologists that better work in a generally neglected portion of an ocular examination can and should be done. This is best described by the author: "Had angioscotoma no other value in a diagnostic sense, its study would still be justified, because it provides a topographical outline which not only helps in the localization of various processes but also supplies a guide to show whether this or that area has been covered in a particular study. If one has succeeded in outlining a few vessel stumps about a blind spot, it is a guaranty that the blind-spot has been carefully and reliably mapped."

All ophthalmologists should own and study this book so that desired additional data will be available in the near future. The author is to be complimented for a work well done.

W. I. LILLIE, M.D.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Opethalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. Stewart Duke-Elder, 59 Harley St., London, W. 1.
Secretary: Dr. Thomasina Belt, 13 Mitchell Ave., Jesmond, Newcastle-on-Tyne.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the Secretary, Dr. Mohammed Khalil.

Ophthalmological Society of the United Kingdom

President: Mr. T. Harrison Butler, Leamington.
Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

OXFORD OPHTHALMOLOGICAL CONGRESS

Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 1939.

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President: Dr. Arieh Feigenbaum, Abyssinian Str. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel-Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Malcolm Hepburn, 111 Harley St., London, W. 1, England.
Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö.

TEL-AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Allenby Str., Tel-Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik Str., Tel-Aviv, Palestine.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung, China.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. S. Judd Beach, 704 Congress St., Portland, Maine.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: St. Louis. Time: May 15-19, 1939.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 8-14, 1939.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Frederick Tooke, 1482 Mountain St., Montreal, Canada.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: Hot Springs, Va.

CANADIAN OPHTHALMOLOGICAL SOCIETY

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal.
Secretary-Treasurer: Dr. J. A. MacMillan, 1410 Stanley St., Montreal.
Place: Montreal. Time: June 19-23, 1939.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.

SECTIONAL

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. O. Ebert, 104 Main St., Oshkosh, Wis.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.
Place: Oshkosh. Time: May 1939.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. F. C. Cordes, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: San Francisco. Time: June 19-22, 1939.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. L. Goss, Cobb Bldg., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. L. J. Friend, 425 E. Grand Ave., Beloit, Wis.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501–7th St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Don M. Howell, Alma, Mich.
Secretary-Treasurer: Dr. Louis D. Gomon, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. R. A. Kelly, 304 N. Main St., Mitchell, S. D.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. Carl Wencke, Battle Creek.
Secretary-Treasurer: Dr. A. K. Zinn, Battle Creek.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. M. Harris, Johnstown.
Secretary-Treasurer: Dr. C. Wearne Beals, 41 N. Brady St., DuBois.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: Capitol Life Bldg., Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. William M. Good, 63 Center St., Waterbury.
Secretary-Treasurer: Dr. S. J. Silverberg, 201 Park St., New Haven.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Grady E. Clay, 384 Peachtree St., N. E., Atlanta.
Secretary-Treasurer: Dr. J. Mason Baird, 511 Medical Arts Bldg., Atlanta.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. W. Rutherford, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: First Wednesday in April.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. H. Lamb, American Bank Bldg., Davenport.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.
Place: Davenport.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. Francis E. Le Jeune, 632 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Place: Gulfport, Miss. Time: May 8, 1939.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. B. Fralick, 201 S. Main St., Ann Arbor.
Secretary: Dr. O. McGillicuddy, 124 W. Allegan St., Lansing.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. Frank N. Knapp, 318 W. Superior St., Duluth.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Roy Grigg, Bozeman.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Norman W. Burritt, 30 Beechwood Rd., Summit.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: Atlantic City. Time: June 1939.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Secretary: Dr. Chester C. Cott, 333 Linwood Ave., Buffalo.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. N. A. Youngs, 322 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516-6th St., Valley City.
Place: Fargo. Time: May 1939.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. O. Clement, 406 State St., Salem.
Secretary-Treasurer: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. S. B. Fishburne, 1430 Marion St., Columbia.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.
Place: Columbia. Time: Nov. 1, 1938.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Kate Savage Zerfoss, 165-8th Ave., N., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 805 Medical Arts Bldg., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.
Secretary-Treasurer: Dr. M. H. Williams, 30½ Franklin Rd., S. W., Roanoke.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Andrew Rados, 31 Lincoln Park, Newark.
Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron, Ohio.
Secretary-Treasurer: Dr. C. R. Anderson, 106 S. Main St., Akron, Ohio.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. J. Mason Baird, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. Fred M. Reese, 6 E. Eager St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. E. Clifford Place, 59 Livingston St., Brooklyn.
Secretary-Treasurer: Dr. Frank Mallon, 1135 Park Pl., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Georgiana Dvorak-Theobald, 715 Lake St., Oak Park, Ill.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Ophthalmological Club

Chairman: Dr. Paul Moore, Republic Bldg., Cleveland.
Secretary: Dr. G. Leslie Miller, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Hugh G. Beatty, 150 E. Broad St., Columbus, Ohio.
Secretary-Treasurer: Dr. W. A. Stoutenborough, 21 E. State St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. Edgar G. Mathis, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Lester H. Quinn, 4105 Live Oak, Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Arthur S. Hale, 1609 Eaton Tower, Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. A. Gough, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 806 Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Louis Daily, 1215 Walker Ave., Houston, Texas.
Secretary: Dr. Herbert H. Harris, 1004 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. J. K. Leasure, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to June.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. E. N. Robertson, Concordia, Kan.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Paul Nilsson, 211 Cherry Ave., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Clifford B. Walker, 427 W. 5th St., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Gaylord C. Hall, Brown Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Charles K. Beck, Starks Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Earle Breeding, 1801 I St., N. W., Washington.
Secretary: Dr. Elmer Shepherd, 1606–20th St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. John E. Mulsow, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

MINNEAPOLIS OPHTHALMOLOGICAL SOCIETY

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman: Dr. P. H. Kilbourne, Fidelity Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., monthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. L. de G. Joubert, 690 Dunlop St., Montreal, Canada.
Secretary: Dr. K. B. Johnston, 1509 Sherbrooke St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Guy Maness, 119–7th Ave., Nashville, Tenn.
Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. E. G. Walls, 619 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Percy Fridenberg, 38 W. 59th St., New York.
Secretary: Dr. David Alperin, 889 Park Place, Brooklyn.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. Philip Romonek, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Ophthalmological Society of the University of Pittsburgh

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Time: Second Monday in November, January, March and May.

Passaic-Bergen Ophthalmological Club

President: Dr. R. N. Berke, 430 Union St., Hackensack, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Walter I. Lillie, 255 S. 17th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward Stieren, Union Trust Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, 351-5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. N. H. Turner, 200 E. Franklin St., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. Roy E. Mason, Frisco Bldg., St. Louis.
Secretary: Dr. Leslie Charles Drews, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Russell Fletcher, 490 Post St., San Francisco.
Secretary: Dr. Avery Morley Hicks, 490 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. R. Kirkpatrick, 6th and Walnut Sts., Texarkana, Ark.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. P. B. Greene, 422 Riverside Ave., Spokane, Wash.
Secretary: Dr. O. M. Rott, 421 Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. James F. Cahill, 428 S. Salina St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. A. Lloyd Morgan, 170 St. George St., Toronto, Canada.
Secretary: Dr. W. R. F. Luke, 170 St. George St., Toronto, Canada.
Place: Academy of Medicine, 13 Queen's Pk. Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 I St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.

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Zona: See Herpes zoster

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